VOLUME LX SEPTEMBER, 1950 NUMBER 9

THE LARYNGOSCOPE

FOUNDED IN 1896
BY
MAX A. GOLDSTEIN M.D.

PUBLISHED BY
THE LARYNGOSCOPE
640 SOUTH KINGSHIGHWAY ST. LOUIS (10), MO., U.S.A.

NOTICE TO CONTRIBUTORS

THE LARYNGOSCOPE reserves the right of exclusive publication of all articles submitted. This does not preclude their publication in Transactions of the various Societies.

Manuscripts should be typewritten, double spaced, on one side of paper only and with sufficient margins to allow for corrections.

References should be complete: author's surname, initials, title of article, journal, volume, page, month, year.

Six illustrations will be furnished for each article without cost to author. Authors will please limit illustrations to six or assume the expense of additional illustrations.

Proofs will be submitted to authors for corrections. If these are not returned, articles will be published as corrected in this office.

Reprints will be furnished at the following prices:

WITHOUT COVER

	250 Copies	500 Copies	1000 Copies	2000 Copies
Four Pages	$10.25	$12.00	$15.50	$24.00
Eight Pages	20.75	25.75	35.00	52.25
Twelve Pages	31.00	40.00	57.00	89.00
Sixteen Pages	40.50	49.00	65.50	98.00
Twenty Pages	48.00	58.75	79.50	121.00
Twenty-four Pages	58.00	70.50	95.00	143.50
Thirty-two Pages	76.75	91.25	121.25	179.00

WITH COVER

Four Pages	$19.00	$23.75	$33.25	$53.25
Eight Pages	29.50	37.50	52.75	81.50
Twelve Pages	39.75	51.75	74.75	118.25
Sixteen Pages	49.25	60.75	83.25	127.25
Twenty Pages	56.75	70.50	97.25	150.25
Twenty-four Pages	66.75	82.25	112.75	172.75
Thirty-two Pages	85.50	103.00	139.00	208.25

Express charges to be paid by consignee.

THE
LARYNGOSCOPE.

VOL. LX SEPTEMBER, 1950. No. 9

RHINOLOGY IN CHILDREN; RESUME OF AND COMMENTS ON THE LITERATURE FOR 1949.

D. E. S. WISHART, M.D.,
Toronto, Canada.

In the journals devoted to otolaryngology the articles referring to rhinology in children continue to shrink, as will be seen from the following resumé which follows the pattern established in previous years.

GENERAL ARTICLES ON RHINOLOGY.

Furstenberg[1] with his usual clarity and sanity justifies another review of therapeutic principles and makes a plea for their more thoughtful and sounder application in a paper on antibiotics in the treatment of diseases of the ear, nose and throat. The paper should be read in its entirety. Certain of his conclusions follow.

Nearly all the acute infections of the ears, nose, sinuses and throat respond in a gratifying manner to the parenteral administration of penicillin. The isolation and identification of the causative organisms in every case of infection is an idealistic pronouncement but hardly feasible and practicable in everyday practice. When, however, a definite clinical response to the antibiotic is not promptly observed, the organism must be identified and its sensitivity determined. No other course will permit the rationalization of specific therapy.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, July 26, 1950.

When dealing with meningitis where time is an essential factor, delay for the identification of the organism can hardly be advocated. There can be no compromise with the prompt institution of therapy. Antibiotics of proven values . . . should be employed in maximum dosage in conjunction with chemotherapy.

Penicillin, to be effective, must come in actual contact with the organism and maintain a sustained contact. The difficulty of applying this principle to the upper air passages is accountable, perhaps, for the disappointing results observed from the popular methods of topical administration. Penicillin aerosol is of little value in the treatment of the lower respiratory infections. It is doubtful that this technique of local application accomplishes more or even as much as the intramuscular injection of the antibiotic.

A dense capsule surrounding a chronic abscess in the neck is a barrier to penicillin therapy; likewise, necrotic tissue within the abscess renders the lesion resistant to the antibiotic. Similar factors which influence adversely the effectiveness of penicillin are found in chronic suppurative otitis media and mastoiditis, osteomyelitis of the calvarium and facial bones, and chronic nasal accessory sinus disease. These pathological entities are not cured by any antibiotic in our present therapeutic armamentarium, either when used systemically, locally, or by both methods.

An admonition seems timely in regard to the possible injurious effects of penicillin in the absence of infection. The normal basic flora of the throat may be altered to include harmful organisms. It is to be emphasized also that the prolonged administration of the antibiotic may produce resistant organisms which fail to respond to penicillin therapy at some subsequent time when the antibiotic is sorely needed. He has a staff member under his observation who has received intensive penicillin therapy for a chronic maxillary sinusitis. The treatment has been futile. More perturbing, however, is the fact that he harbors a pneumococcus in his antrum which is resistant to penicillin. This raises the question, has this

patient lost an important defense against the possible future development of pneumococcic complication?

Enders[2] reviews briefly those viruses pathogenic for man which, during the course of the disease they incite, may be present in the mouth, pharynx and adjacent structures and then considers in more detail our present knowledge of three virus diseases: acute herpetic gingivostomatitis, stomatitis and diarrhea of infants, and mumps.

He finds that two misconceptions regarding viruses are somewhat widespread: *1.* that laboratories equipped for the study of viruses are prepared to attempt the isolation of virus or to carry out diagnostic serologic tests on any specimen which may be submitted for "virus studies"; and *2.* that a tentative diagnosis of "virus infection" is appropriate in conditions which appear to be infectious but in which no etiologic agent can be identified on the basis of either clinical or laboratory data.

Seventeen virus diseases are listed in which the agent may be present in oropharynx or nasal passages. Although in none of these is it possible by laboratory methods to confirm or establish the diagnosis their impracticability in the routine diagnosis of a disease as important as nonparalytic poliomyelitis as well as their complete deficiency in most of the common viral diseases of childhood must be regarded as unsatisfactory.

When the laboratory diagnosis can be made it is based either upon the isolation of the virus in a susceptible animal or upon the demonstration of the development of specific antibody during the acute phase and early convalescence. Even with those viruses that are most easily propagated in the laboratory, such as influenza, mumps and herpes simplex, the procedures are time-consuming and expensive. Accordingly, this mode of diagnosis in most instances should be employed only under special circumstances, as in the determination of the prevailing agent in epidemics or where the clinical data strongly suggest the nature of the infection.

He also points out that the serological findings nearly always provide a diagnosis only in retrospect, since recovery frequently occurs before they can be obtained.

The Journal of the American Medical Association[3] made the following comment upon the treatment of the common cold:

Authoritative medical opinion supports the view that no substance or combination of substances available at present can be relied upon to prevent or cure the common cold. Recent surveys of products which now appear on the market reveal that many of them make claims involving the treatment or prevention of colds which are not justified by valid evidence. Advertisers exaggerate the effects which the medicaments will have upon symptoms; nevertheless, the public will continue to spend huge sums of money unnecessarily on obsolete, illogical remedies, such as rubbing remedies, salves and ointments, until it is educated to the foolishness of such practice. A number of simple agents and measures exist which families can be taught to employ for temporary relief and from which little harm will result.

The use of nose drops and inhalants probably ranks first among measures taken by the public to relieve colds. Nasal vasoconstrictors afford symptomatic relief for varying periods but are not curative. Liquid nasal medicaments which function on a rational physiologic basis are compatible with ciliary activity, do not vary greatly in their pH (5.5 to 6.5) from that of normal nasal secretions, are isotonic and are noninjurious and nontoxic. Prolonged and excessive use of nose drops and volatile inhalers occasionally results in secondary rebound congestion of the nasal mucous membranes. Prolonged administration of silver salts can produce argyrosis, while the continued use of nasal preparations containing mineral oil sometimes leads to the development of lipid pneumonia.

Although popular with the public, gargling has little therapeutic utility. Ordinarily, gargles do not provide medication behind the anterior pillars of the fauces. A number of investigators find that gargled materials will not pass beyond the

third molar tooth, thereby reaching neither the tonsillar nor pharyngeal areas, because the act of gargling produces a restrictive influence. In most cases of acute, uncomplicated pharyngitis, a reasonable amount of relief may be obtained by irrigating the throat with hot, isotonic solution of sodium chloride by means of syringes or modifications of a douching apparatus.

Acetylsalicylic acid does not have any influence upon the infectious process of the common cold. Symptomatic relief of headache, fever and muscle pain is, nevertheless, of value as long as the drug does not disguise the need for more vigorous treatment. A false sense of improvement may occasionally occur, thereby permitting the patient to resume activity too soon. Numerous cough mixtures to which laymen resort without the advice of a physician are of doubtful value. Expectorants or respiratory sedatives are best prescribed by the physician, as the preparation of the cough mixture should be governed by the type of cough and the age of the patient.

From time to time claims are advanced for special diets for either the prevention or the cure of colds. The dietary measures most frequently recommended for the prevention of colds include a high protein diet, a diet low in carbohydrates and large quantities of citrus foods, presumably to establish "alkalinization." None of these theories is supported by carefully controlled studies. The supplying of extra vitamins and other food elements to the person who does not show signs of deficiency disease does not protect him against colds. Procedures which have as their objective the conditioning of the body to sudden changes in external temperature of the skin, such as cold and hot shower baths, do not lower the incidence of the common cold. The scientific evidence against the value of oral cold vaccines is overwhelming.

The treatment of the common cold consists mainly in the relief of symptoms as they arise. Treatment should never be standardized but should be suited to the needs of each patient. During the earliest stages of the common cold the prime objective is to supply moisture by means of steam inhalations to

the stricken upper respiratory passages. Alcohol has been utilized for generations to abort impending colds or to treat them. In reasonable doses it causes peripheral vasodilatation and re-establishes circulation in chilled cutaneous and mucosal surfaces. Little attention need be paid to fluids in the average uncomplicated cold; it is probably best to let thirst to a great extent determine the fluid intake. Cathartics and laxatives, long employed as home remedies for colds, do not have particular value; their excessive use may lead to dehydration. Rest in bed, especially if fever is present, diminishes the severity of the common cold, limits its spread to others and reduces the frequency of complications. It has stood the rigid test of time as a most sane and effective measure.

The Council on Pharmacy and Chemistry of the American Medical Association[4] at a recent meeting (Autumn, 1949) warned against the indiscriminate use of antihistaminic substances which are now being promoted for the prevention of colds and even for the treatment of those suffering from colds. The Council, while recognizing that data have been accumulated relative to such uses, is not convinced that they are sufficient to warrant the positive statements that are being made. The Council warns that instances have been reported of users of these drugs becoming drowsy and even falling asleep while at work and, in occasional cases, while driving cars or operating machinery. A review of the present status of these products will be prepared so that physicians who prescribe the drugs may be aware of their possibilities. In the meantime the Council declares that experience with these substances is insufficient to permit knowledge of whether they are harmless when used over long periods of time; furthermore, the amounts taken in persistent colds may exceed what has been established as normally safe.

TREATMENT OF ACCESSORY SINUS DISEASE.

De Boissière[5] very rightly observes that it is in young children suffering from sinusitis that one's therapeutic ingenuity is taxed to the limit. He has treated 350 cases and used 75 other cases as controls. In no case was it necessary to resort

to operation. Freedom from the concomitant symptoms was early and sustained, and no harmful results to the mucous membrane of the nose were noted. Recurrences have been infrequent, although a few have occured, but in these symptoms have been less severe than at the initial infection.

The methods he has found most satisfactory in the elimination of sinus infection are twofold: *1.* X-radiation; and *2.* Proetz displacement with penicillin 1:200 in physiological saline; in addition, the instillation of a vasoconstrictor in an aqueous physiological solution and instilled by the Parkinson's method.

Puffiness under the eyes in children is often an accompaniment of sinusitis. This fact, however, is not as widely recognized as it should be. A correspondent wrote to *The Journal of the American Medical Association* inquiring into the significance of this sign and received a reply[6] which is abbreviated as follows:

"In the diagnosis of this group of children, whose main symptom is a puffiness of the face, particularly around the eyes, there are a number of considerations which might be of aid. One might ask whether this puffiness is hereditary and whether other members of the family evidence the same symptom. This type of puffiness, of course, would not be outgrown."

While the author of this inquiry may be describing a new clinical entity, it is more probable that the puffiness noted in children in this age group is due to respiratory infections and secondary anemia. These children outgrow this condition. It is a common observation that some small children, particularly those from three to eight years of age, seem to suffer an interminable number of respiratory infections, particularly during the winter months while they are at school. These infections produce a secondary anemia of a moderate nature, and a constant inflammation of the throat and nasal passages makes their faces appear puffy under the eyes. At about eight years of age or thereafter, these children seem to acquire

an increased immunity or resistance; they have less frequent respiratory infections and outgrow puffiness of the face.

That the spirit of the children is satisfactory, as are their school records, is an important differential point, for were any severe systemic illness causing the puffiness of the face as described, the children would act chronically ill and their school record would suffer.

Further diagnostic measures do not seem to be called for as the condition is self-limited.

The above answer evoked the following abridged comment:[7]

The answer to the inquiry regarding puffiness under eyes in children did not mention chronic sinusitis, the probable cause in most cases in the northern part of the United States. This is particularly true in the absence of positive urinary findings; furthermore, the associated symptoms mentioned, such as anorexia, secondary anemia and frequent colds, are common in chronic sinusitis.

Colds and puffs under the eyes were the rule in cold-susceptible children in the chest clinic and in children with rheumatic fever. In 100 rheumatic children, all of whom had chronic sinusitis, the correspondent and his co-workers had reported that 91 per cent had circles under the eyes and many had puffiness as well, particularly upon arising. The sinuses fill up during recumbency, particularly if prolonged, and produce a congestion under the eyes and beside the nose.

The Journal of the American Medical Association[8] shortly described the Proetz treatment for sinusitis for an inquiring correspondent as follows:

The Proetz displacement therapy for sinusitis consists of placing the patient's head in a hyperextended position and then filling the nasal passages with isotonic sodium chloride solution containing a dilute shrinking agent, such as 0.12 per cent phenylephrine hydrochloride or a 0.25 per cent ephedrine, and applying suction while the patient closes his palate. The suction should be intermittent in order gradually to evacuate

the air and the secretion from the sinuses, thus allowing the solution to enter.

The treatment is of value in certain cases of chronic sinusitis. It is contraindicated in acute sinusitis, where it may result in spreading the infection from infected sinuses to uninfected sinuses.

Hynes[9] hopes to prove that penicillin aerosol has a place in the treatment of sinusitis. The apparatus he uses is that of Barach. The technique consists of having the patient take three or four breaths of the penicillin vapor, followed by suction (60 mm. of mercury), alternating in this way until the penicillin is entirely vaporized. A small amount of saline is then added to obtain as much of the penicillin left in the apparatus as possible. The principle involved is to obtain a negative pressure in the sinuses so that the aerosol will enter when inhaled under slight pressure. Altogether, 212 patients were treated and of these 58 were classified as clinical cures. A number of his very young patients have been treated. Most of these had been treated previously for "sinus," and had had tonsillectomy and/or adenoidectomy. Many had had a few (two to 10 tests) for "allergy." Outside of two entirely uncooperative children, every one showed marked improvement with occasional recurrence.

Winborn[10] discusses frontal sinus infections, their complications and management, and reports five cases, two of which were in children 15 and 13 years of age, respectively. He contends that extensive surgery is not necessary and that mortality is low in frontal sinus complications when these are treated conservatively in conjunction with penicillin and sulfonamides. When the quiescent stage is reached following a complication, a chronic frontal sinus should be exenterated by an external approach in order to avoid a possible recurrence.

Kler[11] states that abscess of the frontal lobe is a rare complication of sinusitis in general and an extremely rare complication of ethmoiditis in particular. In a paper on that complication he gives a description of its occurrence in a five-year-old child. The determining factors in the diagnosis were

the history of an infection of the upper respiratory tract, followed by the obvious presence of a sinal infection, a periorbital abscess and signs of meningeal irritation and increased intracranial pressure. Operation revealed necrotic bone, an extradural abscess and a fistulous tract to a subcortical abscess. Recovery.

Banham[12] reports a case of osteomyelitis of the maxilla in a child of two and one-half years. Complete recovery, without the formation of sequestra, followed conservative surgery and large doses of penicillin.

About one week after damage to her left cheek from a fall her left lower eyelid was swollen, there was purulent discharge from the left side of her nose and from the sockets of two teeth which had been removed, and there was swelling of the hard palate on the left side. In spite of penicillin therapy her condition did not improve and the abscess of the hard palate was incised, the orbital abscess drained by an incision along the lower orbital margin and an intranasal antrostomy was performed. Penicillin was given three hourly for 21 days to a total of 11,000,000 units.

ALLERGY.

Grove[13] after 18 years of close contact with the allergy clinic of the Roosevelt Hospital in New York City is convinced that the importance of the disease of the sinuses in the treatment of allergic manifestations is too often underevaluated by the allergists and also by the otolaryngologists. He deprecates the pessimism of the latter and undertakes by three tables and the summary of six very complete studies of the results of his own sinus surgery in asthmatic patients to prove that excellent results can be produced by careful intelligent handling of disease in the paranasal sinuses. The cases analyzed were followed postoperatively for one-half to 17 years. The number of children in his study is not stated.

Two of his observations are noted: he does not believe that the simple washing of an antrum and finding it clear, and the report of a negative culture of the return flow indicates a

normal or noninfective sinus; he uses a 25 per cent colloidal solution of thorium dioxide diluted one or two times for the demonstration by X-ray of the condition of an antrum and finds the shadows less dense and the margins and backgrounds of the sinus more clearly delineated than when lipiodol is used.

Edwards[14] presents certain aspects of a series of 143 cases of perennial allergic rhinitis and asthma. In 122 cases definite information as to the age of onset shows that this is essentially a malady commencing in middle childhood. Only two cases began under one year of age and seven under two years. In about one-half of the cases the onset dated in the age group from five to 10 years. All the patients were skin tested. Chocolate gave the greatest number of positive reactions to foods. The position of corn on this list — fourth — is of interest because it is so often used as the cereal basis for elimination diets.

Removal of tonsils and adenoids was noted in 44 cases. No relationship between the severity of the allergic manifestations and the presence or absence of the tonsils was apparent in these cases. He quotes an author as stating that tonsillectomy usually aggravates the asthma, and many children actually date their asthma from a tonsil operation; but he is convinced that children with respiratory allergy stand ether anesthesia well and feels that if tonsils are chronically diseased they should be removed, and other foci of infection cleared up in the program for improving the patient's general health.

He particularly refers to hypodermic desensitization using a mixed inhalant allergen and draws attention to the importance of the psychological management of these cases.

Levin and Moss[15] have evaluated clinically Thephorin, a new antihistaminic drug, differing structurally from previous antihistaminics, in 109 allergic children. It was found to be very effective for symptomatic relief. It produced side reactions in 20 per cent of their cases. Reactions were seldom severe enough (8 per cent) to discontinue use of the drug. Thepho-

rin has a mild sedative effect in children in contrast to the stimulating effect seen in adult patients.

Eosinophilia is so frequently thought to be unmistakable evidence of the allergic state that a statement by *The Journal of the American Medical Association* on the significance of eosinophilia[16] is worth noting.

In general, eosinophilia is not present unless the eosinophil count is greater than 6 per cent or the total number of eosinophiles is more than 600 per cmm. A number of disorders may produce mild eosinophilia. These disorders are practically all allergic or parasitic or consist of involvement of the bone marrow. If the eosinophile determination is greater than 20 percept, the diagnostic field is somewhat restricted. Trichinosis or arsenic poisoning are most commonly associated with such high percentages, but eosinophilic leucemia, periarteritis nodosa, or lymphosarcoma may show such percentages. An eosinophilia of less than 20 per cent may indicate any one of a great number of parasitic infestations, allergic states or even bone marrow involvement.

EFFECTS OF DRUGS.

Terrell and Hoar[17] report the successful treatment with streptomycin of four young infants suffering from hemophilus influenzae laryngotracheobronchitis. The lack of clinical characteristics in young infants which would identify the etiology of the disease is shown. It is suggested that streptomycin be given immediately to all cases of laryngotracheobronchitis occurring in young infants and continued until the throat culture reveals a nonsusceptible organism.

Amethocaine hydrochloride as a surface analgesic is increasing in popularity in Great Britain. American readers will more readily recognize this drug under one of the following names: "pantocaine," "pontocaine," "anethaine," "tetracaine." Laryngologists use it in the nose and throat and preparatory to bronchography and bronchoscopy.

Its advantages are: it is resistant to boiling; it is stable in solution; it is lethal to all nonsporing organisms; it is 10 times

more potent than procaine or cocaine; and it is relatively quick in achieving its action and provides prolonged analgesia. Although the onset of analgesia is slower than with cocaine, the effect is more persistent.

Its disadvantages are: it penetrates the tissues more rapidly than procaine and does not produce the vasoconstrictive action of cocaine. It is, therefore, essential to use it in conjunction with adrenalin, which reduces its toxicity by one-fifth.

Jackson[18] reports two toxic reactions in the use of this drug as an analgesic for bronchoscopy. In reviewing the literature he finds 12 reported deaths and many cases of severe constitutional disturbance. He shows that an adequate sensitivity test is difficult and makes valuable suggestions for the prevention and treatment of toxic reactions. His paper is recommended to all those who are in the habit of using pontocaine.

PHYSIOLOGY.

Fabricant and Perlstein[19] had an unusual opportunity to study the hydrogen-ion concentration of nasal secretion *in situ* in a group of 13 newborn infants ranging from two to eight days in age. Three consecutive readings were taken at one-half-minute intervals. For the 12 newborn infants who cried, the hydrogen-ion concentration of the nasal secretion was found to range from 6.7 to 7.2.

NASAL HEMORRHAGE.

Ogura and Senturia[20] present a long, well illustrated and tabulated study of the incidence, cause and control of nasal bleeding seen in a large hospital center where both children and adults were observed. In 14 years 48 children were hospitalized for epistaxis. Epistaxis was noted present in 50 per cent of children with active rheumatic fever or inactive rheumatic heart disease. Epistaxis in children was always anterior and septal.

TONSILS.

Anderson[21] writes for general practitioners an unusually practical paper on the acute throat. The article should be read in its entirety, but some of his remarks on treatment which aroused comment require mention: *Antitoxin*—Although in many cases treatment will be guided by the results of the bacteriological examination, it is often necessary (and in a suspected case of diphtheria essential) to start treatment before the results of such examination are known. The dose of diphtheria antitoxin ranges from 5,000 to 100,000 units. Where the disease is suspected an intramuscular dose of 8,000 units will suffice. For the late, severe case when hospital admission cannot be immediately secured a dose of the order of 50,000 units will be required, and in such an event intravenous administration is particularly desirable. Such cases are best treated in hospital, however, and the practioner will find that most fever hospitals are anxious to admit them even when the diagnosis is unconfirmed or in doubt. *Penicillin*—For the coccal infections and for Vincent's angina penicillin is the treatment of choice. It should be realized that the need is to secure an adequate local concentration of the antibiotic. The condition must not be thought of as a mere surface infection. Local therapy by lozenges and so forth is, therefore, completely inadequate, and systemic administration in high dosage is indicated. If the aqueous solution is used a four-hourly dose of 100,000 units should be injected intramuscularly; procaine penicillin may be employed to advantage here, and two or three injections of 500,000 units during the first 24 hours may be followed by a lower dosage for the remainder of the time. For all throat infections due to susceptible organisms treatment must be continued for seven days to secure the best results.

Although there is no specific treatment for infectious mononucleosis the throat infection is usually due either to streptococcus pyogenes or Vincent's infection, and penicillin will prove effective. In agranulocytic angina, also, the administration of penicillin has greatly improved the outlook. Pentnucleotide should be injected intramuscularly. During the

first 24 hours three or four injections of 5 to 10 ml. may be given, and thereafter the dosage will depend upon clinical response. The administration of the antihistamine drugs might be considered, but the results have not been encouraging. In severe cases repeated small blood transfusions are valuable. The diet should be plain and contain no flavorings or adulterants, and care should be taken to avoid the use of any chemical which might have a sensitizing action. *Sulfonamides*—The administration of sulfonamides has been intentionally omitted from the treatment of the acute throat. The efficacy of these drugs has never been convincingly demonstrated in such conditions. Since they are undoubtedly toxic materials, carrying appreciable risks of sensitization, their administration is inadvisable, and in certain conditions may even be dangerous.

Finally, it must be pointed out that many mild throat infections clear rapidly and effectively without specific treatment. Gargling is so frequently indulged in by the patient before seeking advice that its continuation may be advised for its effect in cleansing the mouth. For those adults who can gargle, warm water with glycerin of thymol may also prove soothing to the throat. When pain is more severe the application of poultices to the neck, inhalations of friar's balsam, and, of particular value, the administration of aspirin powder, which is allowed to melt slowly in the mouth before swallowing—such measures are often adequate to secure rapid relief. The hasty rush for penicillin as a panacea for all cases is to be deprecated. It may do the patient little harm, but it can induce in the physician an easy optimism which may eventually cloud his whole judgment.

Various correspondents criticized Anderson's views on the sulfonamides, calling forth the following reply:[22]

"I was, of course, aware that sulfonamides were widely regarded as efficacious in tonsillitis; nevertheless, it is necessary to be clear about what the sulfonamides are expected to do. I imagine I am not exaggerating if I say that of every 100 sore throats which are severe enough to make the patient

seek medical advice at least 70 of them will subside, by means of the hosts' natural defenses, within a matter of 24 to 48 hours. I am not satisfied from my own clinical observation that sulfonamides hasten this process. Further, one of my assistants, Dr. J. O. French, conducted a very carefully controlled investigation of the efficacy of sulfonamides in scarlet fever. She was unable to observe any beneficial effect upon the throat or upon the duration of pyrexia in those given sulfonamide compared with a group of patients (alternate cases) not so treated. When an infection shows a spontaneous tendency to rapid cure in a high proportion of cases one must be wary of making unqualified claims for a certain measure of treatment.

"In the second place, the clinical diagnosis of streptococcal throat makes a further difficulty. From my own clinical observation, supported by adequate bacteriology, I am far from convinced that I can diagnose streptococcal tonsillitis on sight. In a fair proportion of apparently typical cases the flora obtained on a blood-agar plate is little different from the normal, and streptococci are disappointingly absent. As a rule, however, it is not difficult to recognize the effects of streptococcal invasion, and I feel that sulfonamide should be reserved for such cases. It was for this reason that I stressed the importance of examining glands, middle ears and sinuses.

"Finally, I would like to plead for thoughtfulness in the use of all the different chemotherapeutic weapons. Coming to terms with the common pathogenic bacteria is, in this imperfect world, an essential part of growing up. The throat is by long experience well equipped to cope with most of the bacteria that meet it. The occasional mild tonsillitis may be no bad thing if, as a result, our immunity mechanisms are stimulated, for we may thereby be the better equipped to deal with later attacks. The unnecessary aborting of mild illness of no danger to the patient just for the saving of a few hours in the total duration of the infection should not too easily be regarded as a good thing. The experience of the clinician in the fever hospital leaves no doubt about the great increase in sensitization rashes. The experience of the clinician and of the

pathologist leaves no doubt about the great increase in the occurrence of polyarteritis nodosa. I would be foolish to say that there was an absolute correlation between these facts and the known increase in the ingestion of sulfonamides, for, of course, there has been an increase in the ingestion of all kinds of chemicals in recent years. But such facts should make us pause before using sulfonamides routinely for trivial illnesses."

Preston,[23] discussing postoperative tonsil bleeding, gives *1.* a brief resumé of the physiology and dynamics of hemostasis; *2.* comments upon 3,260 cases of tonsillectomy to emphasize certain etiological factors; *3.* a brief survey of the diagnosis and management of hemorrhage; and *4.* a general plan of management of the operation of tonsillectomy designed to reduce postoperative tonsil bleeding.

Williams[24] reports four cases of septicemia in an estimated 30,000 tonsillectomies while he has been in charge of that work at the Bristol Royal Infirmary. That in quite large series of operations so few cases of acute septicemia occur, shows how rare is this complication. He hopes that with modern methods treatment will be more satisfactory.

Alexander and Reynolds[25] state that cerebral complications of operations on the tonsils and adenoids are rare and for that reason report abscess of the left frontal lobe of a diffuse type and without a definite capsule which occurred in a child following that operation. The boy was in normal condition at the time of operation but suddenly developed abnormal symptoms at the fifth postoperative day, death occurring on the fifteenth postoperative day.

This case is of peculiar significance as it followed tonsillectomy and adenoidectomy during the infantile paralysis season and a diagnosis of poliomyelitis was made—a reasonable conclusion considering the neurological findings. The autopsy alone disclosed the true nature of the cause of death. The case suggests the possibility that conditions diagnosed in the past by others as poliomyelitis following tonsillectomy and

adenoidectomy may have been intracranial complications of different origin.

Cook and Munro-Ashman[26] describe the epidemiological and bacteriological findings in an outbreak of streptococcal tonsillitis in a residential school. The outbreak extended over two terms, and 57 out of 340 boys were attacked.

All the early cases were in the same house as the first case, a heavy nasal carrier. A nasal carrier was also associated with each of the three dormitories with the highest attack rates during the time fresh infections were occurring. In most cases, however, the dormitory did not appear to be of special importance in the spread of disease, and the significance of this is discussed.

The early detection and isolation of heavy nasal carriers before widespread dispersal of infection has occurred is emphasized.

IRRADIATION WITH THE NASOPHARYNGEAL APPLICATOR.

Dow[27] reports observations on 74 patients treated with the Army type 50 mg. radium sulfate nasopharyngeal applicator for the destruction of hypertrophic lymphoid tissue in the nasopharynx. One-third of these were children. Forty-one patients were treated by placing the applicator eight and one-half minutes to each side of the nasopharynx: 33 patients were given similar treatments 12 minutes to each side of the nasopharynx. The longer treatment was observed to have removed lymphoid tissue safely and more effectively.

Although in America many reports have been made of radon treatment to the nasopharynx, few have appeared in Great Britain. At the Royal Hospital for Sick Children in Aberdeen this form of therapy has been in operation for about three years. Collins, Thomson and Swindell[28] have made a preliminary report on 41 children who have been treated and adequately followed up.

The nasopharyngeal applicator used by the authors differs considerably from that in common use in America. A glass

capillary tube 3 cm. long, containing 20 mc. of radon is inserted in a platinum tube of wall thickness 0.5 mm. The platinum tube is mounted in a brass holder which can be introduced through the nostril into the nasopharyngeal cavity.

One of these applicators is passed through each nostril in such a way that the radon containers lie parallel to each other and separated from the walls of the nasopharynx by a few millimeters at the nearest point. The applicators are left in this position for one hour.

The platinum wall of the tube is sufficiently thick to absorb the B-rays almost completely, and in consequence the tube is considered a source of pure G-radiation. Two illustrations are provided showing isodose curves superimposed upon radiographs taken in two mutually perpendicular directions of the applicators in position. The illustrations will be considered by Americans as indicating that the authors' applicator must be much more difficult to use than the usual monel metal applicator. The authors calculate the dose delivered in one hour in the neighborhood of the Eustachian cushion as about 300 r. At a depth of 1 cm. in the tissue the dose decreases to 70 r. and at a depth of 2 cm. to 30 r. They point out that the American monel metal tube containing 50 mgm. of radium sulfate in contact with the tissue delivers in six and two-thirds minutes at a depth of 1 mm. 1,520 r.

The chief dangers which may arise from irradiation therapy are:

1. Interference with the centers of ossification. They point out that the field of irradiation is very small and consider this danger likely to arise only if X-ray therapy is used.

2. Irradiation burns leading to scarring and occlusion of the Eustachian tubes. In the dose used by the authors the risk from burning is claimed to be negligible providing no attempt is made to bring the applicator into contact with the Eustachian tube itself.

3. Overdosage which may leave a wide and patulous Eustachian tube as an easy avenue for infection of the middle ear.

4. Reduction of activity of the ciliated epithelium in the nasopharynx.

The authors consider that all these dangers can be avoided by the use of small doses given at widely spaced intervals. They have judged their results more by the symptomatic improvement than by the decrease in the hypertrophy of the lymphoid tissue.

Examination of the postnasal space was made by a small postnasal mirror in the majority of cases. There was no evidence of any radiation burns nor was there any scarring of the nasopharynx. In no case was any atrophic nasopharyngitis observed.

The results obtained from radon application to lymphoid tissue in the nasopharynx are, in their opinion, superior to any that they have achieved with the use of antral lavage, nasal drops, vitamin therapy or any other forms of treatment. Radon therapy caused considerable symptomatic improvement and no harmful effects were observed.

The author of this resumé feels it necessary to comment that in the list of references appended to the article there is not one paper by the Johns Hopkins group who originated this form of treatment and that examination of the nasopharynx of children by postnasal mirror is usually useless for the determination of its condition.

Schulz and Robbins[29] presented before the huge audience attending the fifty-third annual session of the American Academy of Ophthalmology and Otolaryngology the dangers of irradiation of hypertrophied lymphoid tissue of the nasopharynx. These were illustrated by pictures of some devastating results of irradiation of various lesions of the skin which appeared many years after the treatment with doses, in some cases, less than that now advocated for the treatment of hypertrophied lymphoid tissue of the nasopharynx.

Their paper should be most carefully read by all those who are interested in the treatment of the nasopharynx by irradiation. Their summary and conclusions are quoted verbatim.

"With the renewed interest in the application of ionizing radiations to the nasopharynx of children and young adults, it seems wise to re-emphasize the fact that an agent which when properly used may prevent deafness or other serious defects also has the power to do great harm. An attempt has been made to demonstrate this latter fact by citing certain unfortunate occurrences that have followed the use of ionizing radiation in the past. In many of these, changes in normal epithelium which were permanent and progressive followed a radiation dosage that was in the approximate order of the dose now recommended for use in the nasopharynx. The facts to be emphasized, often unknown or forgotten, are that these changes may not appear for many years and that multiple doses are probably more hazardous than a single application. Fortunately, breakdown of atrophic irradiated tissue does not occur very often, and malignant degeneration with even greater rarity; but, for the unhappy individual in whom one or the other does occur, the final picture is frequently worse than the original. If the breakdown of tissue is in a location readily visualized and accessible to plastic surgery, the resulting damage is not irreparable. The nasopharynx, however, is not such a location.

"*Conclusion: 1.* The monel metal nasopharyngeal applicator, while of undenied value in the treatment of hypertrophied lymphoid tissue in certain instances, holds at the same time potential dangers.

"*2.* When it is used in the treatment of benign lesions, particularly in the case of children and young adults, the following suggestions are offered: *a.* The operator should determine by trial on his own arm the erythema dose of the particular applicator he is to use. *b.* No more than an erythema dose should be given without very careful consideration. *c.* The natural urge, when a procedure seems to have promise, to increase the dosage should be avoided. *d.* The method should

not be made a routine procedure or a routine part of an operative procedure. *e.* Careful thought and consideration of each individual case should be given before ionizing radiation is employed."

Robbins and Schulz[30] wrote a second paper emphasizing the dangers of the present vogue of treating hypertrophied lymphoid tissue in the nasopharynx with radium.

Out of a rather large number, a few examples of unfortunate results encountered during the early years of radiation therapy at the Massachusetts General Hospital, the Massachusetts Eye and Ear Infirmary and the Collis P. Huntingdon Memorial Hospital are given to illustrate that which was discovered by the bitter experience of others in the past. Three striking photographs illustrate this statement.

In the early years the radiologist or surgeon was learning the therapeutic possibilities of ionizing radiation. In these cases late radiation necrosis had followed treatment of benign lesions, and many of the changes in normal epithelium, which were progressive and permanent, had resulted from radiation dosage that was approximately the same as the dose recommended for use in the nasopharynx for hypertrophy of lymphoid tissue. *The changes were latent, some not appearing for 10 to 20 years after treatment.*

With the idea in mind that the mistakes of the past may not be repeated in the present, certain precautionary measures are suggested: *1.* that the use of the monel applicator should not be routine; *2.* that for benign lesions in children and young people this treatment should be carried out cautiously and only after careful consideration of each case; *3.* that, except in the selected case, the treatment when given should consist of no more than erythema dose; *4.* that what constitutes an erythema dose should be determined for a given applicator; and *5.* that should no other means of determining the proper dosage for an applicator be available, the operator should estimate it by trial on his own skin.

(The unstated inference was that a dosage of irradiation dangerous for the skin would be equally dangerous to the

ciliated epithelium of the nasopharynx. No reference was made to the research on this subject which had been done in Boston years before.)

Heine[31] reported, in 1936, from the Mosher Laboratory, Massachusetts Eye and Ear Infirmary, a study of the effects of radium upon ciliated epithelium. The article is clearly written and accompanied by eight excellent microphotographs. The summary and conclusions are given verbatim.

"The foregoing paper is in two parts. The first deals with some of the outstanding contributions to the literature within the last five years, pertaining to the effects of drugs upon ciliated epithelium.

"The second part has recorded observations made upon the behavior of ciliated epithelium after exposure to radiation. Three methods of approach to this problem were used; namely, the exposure of hanging drop specimens of ciliated epithelium to the action of radon seeds; second, the exposure of sections of rabbit's trachea to the direct action of Roentgen ray; third, the implantation of a fixed dosage of radium in the nostrils of rabbits and observations upon the histologic specimens made at varying lengths of time after the implantation.

"*Conclusions: 1.* Goblet cells may be another form of ciliated epithelial cells; furthermore, they are probably degenerative, as shown by changes noted in the nasal mucosa of rabbits after exposure to radium.

"*2.* The metabolic rate of the cells evidently was not affected by Roentgen ray when this metabolic rate was measured by changes in the pH of the solutions containing the tissues.

"*3.* It may be possible that some dosages of Roentgen ray are actually stimulating; therefore, there should be a dosage that will produce beneficial results.

"*4.* Ciliated epithelium is not a delicate structure. If one considers that a single dose of unfiltered Roentgen ray of eight erythemas is the maximum for the skin of man, the picture of the severity of a burn produced by a single dose three times that amount, or 24 erythemas, is almost beyond

one's imagination; however, the evidence points toward the fact that these frail-appearing cells do stand such a dose with impunity.

"In searching for an answer to the question, 'Why are these cells so remarkably viable?', I can find no better explanation than the one offered by by Dr. Werner Mueller, namely, that cilia are an ancient structure biologically; that, therefore, in the order of events, they should be the last to be destroyed."

POLIOMYELITIS AND TONSILLECTOMY.

The question whether or not tonsils and adenoids should be removed from children during the months when poliomyelitis is occurring is debated in practically every hospital in the United States and Canada. Yearly articles on this problem have been reviewed here, and the problem is still unsettled.

Aycock[32] criticizes the way the question has been investigated and points out some of the defects of the various analyses in a most thought-provoking article which your editor advises should be read in its entirety.

He believes there is conclusive evidence that the virus of poliomyelitis is widely disseminated and that the paralytic disease is the exceptional manifestation of infection with the virus; and, furthermore, that there are reasons that this exceptional but serious consequence in what is ordinarily an entirely benign and trivial virus infection may be determined not so much by the circumstances of exposure to the virus but by factors which make for susceptibility in a certain few of the many who are exposed.

The articles which steadily appear voice opinions which vary all the way from the point that there is no relationship between tonsillectomy and poliomyelitis to the belief that all surgical procedures on the upper respiratory passages should be suspended during the "poliomyelitis season."

As a matter of fact, the two diametrically opposed points of view seem to take their origin not so much in any divergent evidence as in convictions stemming, on the one hand from a

willingness to put any precautionary measure against poliomyelitis before other medical considerations; and, on the other hand an unwillingness to let anything interfere with convenience, if we are to believe such often heard statements as "getting ready for school" applied to the operation.

From the nature of the situation, with a very sizable proportion of the population experiencing a tonsillectomy at one time or other and only a very small fraction of the population ever suffering an attack and an even smaller fraction suffering the bulbar form of poliomyelitis, any actual association between the two would not be strikingly apparent in even a very large series of tonsillectomies, while it might easily be seen in a much smaller series of cases of poliomyelitis. In other words, one could do many tonsillectomies without encountering such a case, but one would not have to see many cases of poliomyelitis before seeing bulbar cases following the operation by a specific interval.

The evidence, furthermore, usually obtained in any statistical survey may not be at once convincing for the reason that the hazard of the disease in recently tonsillectomized individuals is numerically small, even though much larger than in the population in general. In large surveys, the excess in frequency of recent tonsillectomy in bulbar cases over that in spinal cases and the excess in the frequency of tonsillectomy over a large assortment of other "surgical procedures" when distributed over a period of 60 days amounts to a statistical dilution which makes it not at all impressive. Had these studies gone one step further to test the crucial question whether tonsillectomy preceding bulbar cases was significantly concentrated at around 14 days preceding the disease, the question of a relationship would have been settled.

There is a tendency toward continued study by broad population survey methods which, by their very nature, are not likely to afford an answer to the question which would be any more decisive (because any differences shown by them are likely to be fractional) than are the few detailed studies which are already available — or, better still, than the clinical

experience seen in any sizable number of cases of poliomyelitis as, for example, the group studies by Eley and Flake.

The risk of bulbar poliomyelitis following tonsillectomy *most of the time in most places* is almost nil; but *for short periods of time in a few places* (epidemics of poliomyelitis) it is a risk to be avoided. The risk is determined by the prevalence of the virus in the community.

In order to avoid the confusion of conflicting policies in the same community situation, and perhaps somewhat as a safeguard against any extreme attitudes in either direction, the nose and throat services in a number of the Boston hospitals are making a practice of following a uniform procedure agreed upon by them and based on information and recommendations furnished by the state health department upon any increase in prevalence of the disease in any of the various areas served by the hospitals.

Because there is a sort of "seasonal epidemic" in tonsillectomy which happens to coincide closely with that of poliomyelitis, he recommends that this favorite time for the operation, unless there be some distinct contraindication, be changed from late Summer "before school opens" to early Summer after "school closes" but before poliomyelitis begins.

Wesselhoeft[33] states that the problem of tonsillectomy deals with the question of whether or not this operation — which is usually coupled with the removal of the adenoids — constitutes a real danger to the patient in the incubation period or carrier state of poliomyelitis. The evidence that there is such a danger was presented in dramatic form in the "K" family of Akron. There were six children in this family, and five of these were tonsillectomized on Aug. 22. By Sept. 7, all five of these children had bulbar poliomyelitis, and three of them died. The one who was not operated upon showed no sign of illness, but the virus of poliomyelitis was recovered from the stool. There were only two scattered cases of polio in Akron prior to this family epidemic, and there were only six additional cases in this city during that September. The most probable exposure of this family was four weeks previous to

the tonsillectomies during a visit with relatives in a distant community where poliomyelitis was present. Other cases have been presented where bulbar poliomyelitis followed closely upon tonsillectomy.

The publication of these cases resulted in a ban on tonsillectomies in most hospitals over the country during the season of the year when poliomyelitis is prevalent; namely, the Summer months. I was one of those who, as a consultant in infectious diseases, urged this ruling on the grounds that this operation is elective and could be postponed to the colder months when there was less likelihood of there being poliomyelitis virus in the nasopharynx. Pedersen and others have given us some interesting food for thought on this matter. Last year I revised my opinion on this, recommending that the ideal time for tonsillectomies was immediately after the close of school in June in this part of the country; also, that tonsillectomies could continue through the Summer months providing there were no cases of poliomyelitis being reported from the community and no known possibility of exposure from outside. In Massachusetts last Summer the state board of health through weekly bulletins kept hospitals informed of the situation of poliomyelitis in this state, as well as over the country at large. It would seem that this method offers a satisfactory solution to the tonsillectomy problem; nevertheless, there is always the warning of the "K" family, and regardless of season, you who take the responsibility of performing an elective operation should be aware of the fact that possible exposure at other seasons may occur. In this discussion we have come to realize that other dangers besides poliomyelitis confront this operation, and that to be forced to carry it out when upper respiratory infections are most prevalent is a disadvantage to be seriously considered. That we were carried too far in our zeal to prevent poliomyelitis through a seasonal ban on tonsillectomies is now clear, but this is just one of many errors that have been made in the numerous efforts in fighting this disease. Until we have some quick and practical method of determining the presence of the virus of poliomyelitis in patients on whom tonsillectomy is contem-

plated, we must rely upon epidemiological data as supplied in Massachusetts.

Cunning[34] summarizes data provided for 1948 on tonsillectomy and poliomyelitis in 10 tables which cannot be given here, and deserve study. After 10 years' study on this subject, following over 17,000 poliomyelitis cases and 35,000 tonsillectomy cases, he is still of the same opinion as he was one year ago, namely, that no definite causal relationship between tonsillectomy and poliomyelitis has been established; therefore, he does not believe that tonsillectomy should be postponed indefinitely simply because the Summer months are the months during which poliomyelitis is prevalent. If, however, there is a distinct rise in the poliomyelitis rate, bordering on epidemic proportions, in any community, all elective operations should be postponed.

Until evidence of a conclusive nature is forthcoming, the surgeon should decide whether or not to perform tonsillectomy during the Summer months and whether the dangers of delaying the operation are greater than the still undetermined risk of increasing susceptibility to poliomyelitis.

The Journal of the American Medical Association wrote an editorial[35] commenting on Cunning's report which has been summarized above. The editorial is condensed as follows:

The journal (March 21, 1942) warned that poliomyelitis, particularly the bulbar form, greatly increased in children after tonsillectomy or adenoidectomy in epidemic areas. . . . Since that time avoidance of such operations during epidemics has been almost universally adopted. Recently, otolaryngologists seem inclined to the belief that a causal relationship does not exist between the two and that bulbar poliomyelitis following tonsillectomy is probably purely coincidental. Cunning as a result of a survey of data covering 1947 felt that the operation should not be indefinitely postponed simply because Summer months are the months that poliomyelitis is prevalent. This report was recently reviewed by Faber (given later), who concluded that proofs previously offered for a causal relationship between incidence of poliomyelitis and tonsillectomy

during epidemic times were still valid and that evidence for this had not been refuted by the Cunning survey.

The weight of past and recent evidence favors the existence of such a relationship. As tonsillectomy is an elective operation, which rarely requires immediate surgical attention, the preventive attitude of delay is justified. Until conclusive evidence is presented that such procedures do not increase incidence of poliomyelitis the inadvisability of operation during periods of epidemic poliomyelitis should be emphasized.

Cunning[36] replied to the above editorial, making a correction and a pertinent criticism. He states the editorial failed to mention the 16,643 cases of tonsillectomy performed and carefully followed in 15 different states throughout the country for a period of two months after operation, in none of which did bulbar poliomyelitis develop.

For four years the Committee for the Study of Poliomyelitis and Tonsillectomies of the American Laryngological, Rhinological and Otological Society, Inc., has honestly attempted to learn the truth concerning the relationship between poliomyelitis and tonsillectomies. This committee, a large number of the country's leading epidemiologists and many local health authories, agrees on two main points: *1.* tonsillectomies and adenoidectomies or any other elective operation should not be performed in a community where poliomyelitis (or any other contagious disease) assumes epidemic proportions, and *2.* tonsillectomies and adenoidectomies can with safety be performed in a community where poliomyelitis (or any other contagious disease) does not assume epidemic proportions.

The effort is being made yearly by statistical study to discover the truth regarding any possible relationship between tonsillectomy and poliomyelitis. One of these is in a sense the official report of the American Laryngological, Rhinological and Otological Society, Inc., and a summary of such report has been made each year in this place. Statistics are, however, unsatisfactory masters and may be read in several ways. The report summarized last year received the following criticism by Faber.[37]

In a recent publication, Dr. D. S. Cunning presented the results of a survey conducted by a committee of the American Laryngological, Rhinological and Otological Society, Inc., on poliomyelitis and tonsillectomy for the year 1947 and also described the results of a similar survey for 1946. He summed up his conclusions as follows: "I am more than ever convinced that there is no causal relationship between the two, and that when a bulbar poliomyelitis follows a tonsillectomy it is coincidental."

This dangerous conclusion, which I fear is already being widely accepted by otolaryngologists, should be very carefully weighed in the light of the evidence presented by the present report and of other evidence totally disregarded by it.

As regards the Cunning data, the following points deserve consideration:

1. Twenty-four cases of poliomyelitis in the reported series actually did follow tonsillectomy in 1947 and 62 cases in 1946. Of these, six, or 25 per cent, were bulbar in 1947, and 18, or 29 per cent, in 1946, whereas the incidence of bulbar poliomyelitis in all cases of poliomyelitis covered by the survey was 11.6 per cent in 1947 and 15.4 per cent in 1946; that is, the incidence of bulbar forms was slightly more than twice as high in persons for whom tonsillectomy has been done as in those for whom it has not.

2. Since the compilation by Aycock, in 1942, and the editorial of March 21, 1942, in the journal tonsillectomy has come to be generally avoided during epidemics, and to a smaller extent even during the Summer and Fall months, because of the widespread fear of poliomyelitis that is felt both by physicians and laity; the result of such avoidance would, of course, be to reduce greatly the incidence of post-tonsillectomy cases and thus to increase the proportion of non-post-tonsillectomy cases.

3. The proof of a relationship between tonsillectomy and poliomyelitis is positive in respect to the high proportion of bulbar cases and cannot be blindly disregarded. Thus Ay-

cock's summary shows 170 cases in which poliomyelitis followed tonsillectomy within 30 days (approximately the upper limit of incubation), of which 121, or 71 per cent, were bulbar, whereas in the 30 to 60-day period only 20 per cent were bulbar. The Cunning survey used a 60-day interval, which may explain the lower incidence of bulbar poliomyelitis than in the 30-day reports. The extraordinary report by Krill and Toomey deserves mention. Five children in one family were subjected to tonsillectomy on the same day, and all came down with bulbar poliomyelitis 10 to 12 days later, three of them dying of the disease. Since the Aycock compilation, numerous other reports of post-tonsillectomy poliomyelitis have appeared (including the 86 of the two Cunning reports) and the 17 of Anderson's paper. The latter particularly should not be disregarded, since it offers clear proofs of the relationship under discussion. Anderson obtained data on tonsillectomies performed in Utah in 1943, both before and during the heavy epidemic of that year. Before (July) the outbreak, the incidence of poliomyelitis after tonsillectomy was 0.7 per thousand; in August, when the epidemic began, it was 1.4 per thousand and at the height of the epidemic it was 19.2 per thousand tonsillectomies (these figures have been calculated from the original data). All the post-tonsillectomy cases of poliomyelitis were bulbar. The incidence of poliomyelitis in children in whom tonsillectomy had recently been done was 2.6 times, and of bulbar poliomyelitis 16 times greater than in the general child population.

The Cunning survey is, therefore, in my opinion, a dangerous one, particularly since it is in a sense the official report of the American Laryngological, Rhinological and Otological Society, Inc., and is apt to obtain wide acceptance, at least among specialists in that field, and might lead to the abandonment of what is now generally accepted as an important means of prevention of poliomyelitis. That the author himself still entertains doubts of the correctness of his views is indicated by a remark that follows the conclusion already quoted: "I do not advocate the indiscriminate removal of tonsils during any severe epidemic and have no desire to place our chil-

dren's lives in jeopardy through injudicious surgery. . . ." It is to be hoped that this sentiment will continue to be felt by all physicians who are responsible for deciding on tonsillectomy, especially during epidemics of poliomyelitis.

SURGERY.

Stoner and Freeman[38] believe the rhinologist owes it to the obstetrician, the pediatrician and the general practitioners to emphasize properly the seriousness of congenital posterior choanal occlusion and to recognize it as a possible cause of the symptoms in cases of immediate difficulty in nursing or breathing of the newborn. They describe such a case and illustrate their treatment, using the inner cannula of a No. 2 tracheotomy tube in each side of the nose to keep the surgically made openings patent. The obturators were removed on the seventeenth postoperative day and at the ninth postoperative month both posterior choanal openings were patent.

Sheehan and Swanker[39] write a short, abrupt resumé of the methods of surgically attacking congenital choanal atresia and conclude that while slightly more difficult in some respects, the transpalatine route affords the most satisfactory surgical approach. The operation they advocate is a combination of Ruddy's incision, Neto's fenestration and Blair's treatment of the nasal mucosa and is outlined in nine steps.

Hanckel[40] reviews various methods of dealing with congenital bilateral choanal atresia and describes in detail and with many illustrations a case which required many interferences over a period of four months to cure. Other authors have likewise advocated a transpalatine approach and have suggested that uneventful quick recovery is the rule. This author is to be admired for writing so plainly about the troubles that may be encountered and his paper is recommended to every otolaryngologist who has to deal with this condition.

Dolowitz and Holley[41] review the literature and find that surgical repair of congenital choanal atresia has, in the past, rarely been successful. Failure has almost always been due to secondary scar formation which closes the opening, despite attempts to produce a permanent fistula by means of skin

grafts, prostheses or repeated cauterizations. Even in those instances in which a permanent fistula has been made there has usually not been a return to normal nasal function owing to the fact that no ciliated epithelium lined the new opening.

They describe a new approach to this problem which permits the obstructing bone to be removed by a transpalatine technique and a new nasal passage to be formed which is lined with flaps of ciliated mucous membrane. Seven drawings adequately illustrate the procedure.

One case is reported in which this technique was used. The patient, an eight-year-old child with unilateral congenital choanal atresia, was discharged from hospital eight days after admission and five days after operation. She has experienced entire relief of her nasal complaints since the operation 31 months ago. The emotional and social readjustment following the alleviation of this minor defect was striking.

Shapiro[42] states that in eight of 12 reported cases of cysts on the base of the tongue in infants the diagnosis was made at postmortem examination. In three of the four cases in which the diagnosis was made in a living child simple incision and drainage resulted in complete disappearance of the cyst. In the fourth case, which was really in the body of the tongue, complete incision via the undersurface of the tongue resulted in cure.

The symptoms usually date back to the very earliest infancy and characteristically vary in the degree of severity from time to time. These infants have a peculiar cry and inspiratory stridor. Intermittently they have attacks of complete respiratory obstruction with cyanosis. Death may occur in one of these attacks. Posturing of the patient may give partial relief from the respiratory difficulties.

He reports a case, pictures a postmortem specimen and a diagram of the conditions existing in a case of a cyst at the base of the tongue.

By direct laryngoscopy the diagnosis should be simple. A carefully used tongue depressor or simple palpation may

reveal the presence of the cyst. (The finger may be too large for palpation, the tongue depressor and the laryngoscope may hide the mass. Tongue forceps should be used to pull the tongue forward and the suspected area seen with the use of a head mirror.)

NEW GROWTHS.

Grotts[43] describes minutely a transitional cell carcinoma of the nasopharynx in a two-year-old child. Five weeks before admission a retropharyngeal abscess was diagnosed and drained. She was given three weeks of penicillin therapy but did not improve and lost weight. The posterior nares were completely obstructed with thick mucus; the soft palate was pushed downward and forward by a rather firm mass which protruded into view posteriorly and appeared dark brown, crusted and necrotic. There was marked surrounding edema and cellulitis. Biopsy resulted in the diagnosis of a transitional cell carcinoma of the pharynx. Following this diagnosis 11 deep X-ray treatments of 210 r. each were given to both the left and the right nasopharynx. She died at home 10 weeks after the onset of symptoms. No necropsy was obtained.

BIBLIOGRAPHY.

1. Furstenberg, A. C.: Antibiotics in the Treatment of Diseases of the Ear, Nose and Throat. *Ann. Otol., Rhinol. and Laryngol.*, 5, Mar., 1949.

2. Enders, John F.: Viruses in the Oropharynx and the Nasal Passages. The Laryngoscope, 59:4:372, Apr., 1949.

3. Answers to Queries: Treatment of Common Cold. *Jour. A. M. A.*, 426, Oct., 1949.

4. Current Comment: Claims for Cold Cures Criticized by Council on Pharmacy and Chemistry. *Jour. A. M. A.*, 1059, Dec., 1949.

5. de Boissiere, V.: The Treatment of Chronic Sinusitis in Children. *Canad. Med. Assn. Jour.*, 14, Jan., 1949.

6. Answers to Queries: Puffiness Under Eyes in Children. *Jour. A. M. A.*, 1068, July, 1949.

7. Answers to Queries: Puffiness Under Eyes in Children. *Jour. A. M. A.*, 428, Oct., 1949.

8. Answers to Queries: The Proetz Therapy for Sinusitis. *Jour. A. M. A.*, 500, Oct., 1949.

9. Hynes, F. J.: Penicillin Aerosol Therapy in Sinusitis. *Ann. Otol., Rhinol. and Laryngol.*, 189, Mar., 1949.

10. Winborn, C. D.: Frontal Sinus Infections—Complications and Management. *Ann. Otol., Rhinol. and Laryngol.*, 280, Mar., 1949.

11. KLER, J. H.: Abscess of the Frontal Lobe Secondary to Ethmoiditis. *Arch. Otolaryngol.*, 125, Feb., 1949.

12. BANHAM, T. M.: Osteomyelitis of the Superior Maxilla Treated with Penicillin. *Jour. Laryngol. and Otol.*, 233, Apr., 1949.

13. GROVE, RUSSELL, C.: The Importance of Sinusitis in Allergic Manifestations. THE LARYNGOSCOPE, 59:6:653, June, 1949.

14. EDWARDS, H. E.: Aspects of Perennial Allergic Rhinitis and Asthma in Childhood. *Canad. Med. Assn. Jour.*, 36, July, 1949.

15. LEVIN, S. J., and MOSS, S. S.: Thephorin: Clinical Results in Allergic Children. *Jour. Pediat.*, 616, May, 1949.

16. Answers to Queries: Eosinophilia. *Jour. A. M. A.*, 1034, Dec., 1949.

17. TERRELL, C. O., and HOAR, C. S.: Streptomycin in the Treatment of Hemophilus Influenzae Laryngotracheobronchitis. *Jour. Pediat.*, 139, Feb., 1949.

18. JACKSON, C. A.: Amethocaine Hydrochloride. *Brit. Med. Jour.*, 99, Jan., 1949.

19. FABRICANT, N. D., and PERLSTEIN, M. A.: Hydrogen-ion Concentration of Nasal Secretion *in Situ* in Newborn Infants. *Arch. Otolaryngol.*, 616, Nov., 1949.

20. OGURA, J. H., and SENTURIA, B. H.: Epistaxis. THE LARYNGOSCOPE, 59:7:743, July, 1949.

21. ANDERSON, T.: The Acute Throat. *Brit. Med. Jour.*, 860, Oct., 1949.

22. ANDERSON, T.: The Acute Throat. Correspondence, *Brit. Med. Jour.*, 1179, Nov., 1949.

23. PRESTON, H. GRANT: Postoperative Tonsil Bleeding. Studies of the Etiology, Prevention and Management. THE LARYNGOSCOPE, 59:3:251, Mar., 1949.

24. WILLIAMS, E. WATSON: Septicemia After Tonsil Operations. *Jour. Laryngol. and Otol.*, 115, Mar., 1949.

25. ALEXANDER, S. A., and REYNOLDS, R.: Abscess of the Brain Following Tonsillectomy and Adenoidectomy. *Arch. Otolaryngol.*, 450, Oct., 1949.

26. COOK, G. T., and MUNRO-ASHMAN, D.: Nasal Carriers and Streptococcal Tonsillitis. *Brit. Med. Jour.*, 345, Feb., 1949.

27. DOW, C. H.: Use of Radium in Treatment of Hypertrophic Lymphoid Tissue in the Nasopharynx. *Arch. Otolaryngol.*, 417, Oct., 1949.

28. COLLINS, E. G.; THOMSON, I. S., and SWINDELL, G. E.: Radon Therapy of Nasopharyngeal Lymphoid Hypertrophy in Children. *Jour. Laryngol. and Otol.*, 17, Jan., 1949.

29. SCHULZ, M. D., and ROBBINS, L. L.: Dangers of Irradiation of Hypertrophied Lymphoid Tissue of the Nasopharynx. *Trans. Amer. Acad. Ophthal. and Otolaryngol.*, 243, Jan.-Feb., 1949.

30. ROBBINS, L. L., and SCHULZ, M. D.: Potential Hazards from Radiation Treatment of Hypertrophied Lymphoid Tissue in the Nasopharynx. THE LARYNGOSCOPE, 59:2:147, Feb., 1949.

31. HEINE, L. H.: The Effect of Radiation Upon Ciliated Epithelium. *Ann. Otol., Rhinol. and Otolaryngol.*, 60, Mar., 1936.

32. AYCOCK, W. LLOYD: The Rôle of the Upper Respiratory Mucous Membrane in the Pathogenesis of Poliomyelitis. THE LARYNGOSCOPE, 59:4:335, Apr., 1949.

33. WESSELHOEFT, CONRAD: The Problems of Tonsillectomy and Tracheotomy in Relation to Poliomyelitis. THE LARYNGOSCOPE, 59:7:726, July, 1949.

34. CUNNING, DANIEL S.: Poliomyelitis-Tonsillectomy Survey, 1948. THE LARYNGOSCOPE, 59:5:441, May, 1949.

35. Editorial: Tonsillectomy During Epidemic Poliomyelitis. *Jour. A. M. A.*, 479, June, 1949.

36. Correspondence: Tonsillectomy During Epidemic Poliomyelitis. *Jour. A. M. A.*, 548, Oct., 1949.

37. FABER, H. K.: Tonsillectomy and Poliomyelitis. Correspondence, *Jour. A. M. A.*, 248, Jan., 1949.

38. STONER, L. R., and FREEMAN, M. S.: Congenital Posterior Choanal Occlusion. *Ann. Otol., Rhinol. and Laryngol.*, 226, Mar., 1949.

39. SHEEHAN, J. E., and SWANKER, W. A.: Surgical Repair of Congenital Choanal Atresia. THE LARYNGOSCOPE, 59:12:1320, Dec., 1949.

40. HANCKEL, R. W.: Bilateral Choanal Atresia. *Ann. Otol., Rhinol. and Laryngol.*, 852, Sept., 1949.

41. DOLOWITZ, D. A., and HOLLEY, E. B.: Congenital Choanal Atresia. *Arch. Otolaryngol.*, 587, June, 1949.

42. SHAPIRO, M. J.: Cysts on the Base of the Tongue in Infants. *Ann. Otol., Rhinol. and Laryngol.*, 457, June, 1949.

43. GROTTS, BRUCE F.: Transitional Cell Carcinoma of the Nasopharynx in a Child. THE LARYNGOSCOPE, 59:12:1355, Dec., 1949.

THE PROGRESSION OF IMPAIRED HEARING FOR HIGH TONES DURING CHILDHOOD.*

STACY R. GUILD, Ph.D.,
Baltimore, Md.

INTRODUCTION.

Impaired hearing for high tones only, of the type termed "abrupt" high-tone loss in Guild's method of classifying audiograms (1932), occurs both in adults and in children. Of the 1,700 persons 20 to 69 years included in Ciocco's (1932) study of the hearing of 1,980 individuals, who were randomly selected and whose auditory thresholds for pure tones were detemined audiometrically over the range of frequencies from 32 to 16,384 cycles per second, inclusive, 794 persons had some form of a simple "abrupt" type of loss for high tones, and an additional 231 persons had this type of hearing loss complicated by the presence of a "dip" at some frequency lower than that of the "drop." In other words, Ciocco's report indicates that about three-fifths of all adults have some form or other of an "abrupt" type of impaired hearing for high tones.

The incidence of the "abrupt" type of high-tone loss is much lower in children than in adults. The largest randomly selected group of children that can be used for comparison with Ciocco's data on adults is the group of 1,365 Baltimore school children, aged eight to 13 years, reported by Guild, *et al.*, in 1940.[10] The hearing acuity of these children was tested for the same tonal frequencies as were the adults of Ciocco's study, and in both studies the same method of classification of the audiograms was used. Slightly over 15 per cent of the audiograms of the first hearing tests made of the ears of this group of 1,365 children belong to one or another of the sub-categories of the "abrupt" type of high-tone loss. From these two studies, the incidence of the "abrupt" type

*From the Johns Hopkins University School of Medicine.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 12, 1950.

of hearing impairment for high tones appears to be about four times as great in adults as in children.

CAUSE OF "ABRUPT" TYPE HIGH-TONE LOSS.

In adults whose ears have been studied histologically, an "abrupt" type of hearing impairment for high tones, at least when the loss of normal acuity begins at the frequencies of 8,192, 5,793, 4,096, 2,896 or 2,048 cycles per second, is usually associated with a more or less extensive absence of the organ of Corti in the basal turn of the cochlea.[5] No histologic observations are available on the ears of children with this type of impaired hearing. The idea that the immediate cause of "abrupt" high-tone loss is the same in children as in adults, namely, an absence of the organ of Corti in part of the basal turn of the cochlea, is strengthened by Guild's recent report[9] that in 52 cases of "abrupt" high-tone loss in children, who were re-examined in each case six years or more after the first test had been made, there was not even one instance in which the highest tone that was well heard was of a higher frequency at the last examination than it was at the first examination. The hearing of some ears with this type of loss had become worse, none had improved.

The remote, or true, cause of impaired hearing for high tones, of the "abrupt" type of loss, can be determined with certainty in only a small proportion of the large number of cases that are encountered by otologists and by others interested in hearing. Sometimes there is a definite history of exposure to acoustic trauma of the blast, or detonation, type so severe that the patient's ears "rang" for days or weeks afterward. In such cases, the diagnosis as to the remote cause of the impairment can be made with a high degree of confidence that it is correct, because there is not only good clinical evidence that exposure to the blast type of acoustic trauma often is followed by an "abrupt" type of high-tone loss but also good experimental evidence that the exposure of animal ears to detonation waves causes a histologically demonstrable destruction of the organ of Corti.[6,11,12,13]

When a cleancut history of exposure to acoustic trauma of the blast type cannot be elicited, even by prodding the

patient's memory, decision as to the remote, or true, cause of "abrupt" type high-tone loss is usually uncertain. Most commonly, in such cases, especially when the patient is a child, the hearing impairment is classified as "congenital" and ascribed either to a failure of the basal turn of the cochlea to develop normally or to a toxic damage of this part of the organ of Corti, or of the nerve fibres from it, by some medication received either by the mother before delivery or by the child during early infancy. Naturally it is difficult to prove, or to disprove, the correctness of such a diagnosis; at best it is unsatisfactory to the otologist who makes it, if not to the patient or to his parents. Many otologists frankly recognize such diagnoses as belonging to the "I don't know" category.

The only satisfaction a conscientious otologist has in making such a speculative diagnosis is the certainty that, whatever the true cause of the impaired hearing may have been, he is not neglecting his duty to his patient when he tells him that nothing can be done to restore the hearing for the tonal range affected by the lesion present.

Fortunately for the afflicted persons, hearing for the conversational voice, either spoken or recorded, is seldom seriously affected by an "abrupt" type of hearing impairment for high tones, except when the impairment is of extreme degree (sub-categories C_8 to C_{12} of Guild's classification of audiograms.)[7] When the loss begins at the frequency of 4,096 or of 5,793 cycles, or yet higher, the patient seldom realizes, until his hearing is tested with a pure-tone audiometer, that he has any hearing defect whatever.

Two reasons are responsible for the fact that almost nothing is known about the rate of development or of progression of impaired hearing for high tones, of the "abrupt" type, except in the cases definitely caused by exposure to acoustic trauma. In the first place, because patients with this type of hearing impairment are unaware of the condition, they cannot usually even offer subjective impressions with respect to the time of onset or the rate of progression of the inability to

hear very high pitched tones. In the second place, because otologists have had nothing effective in the form of treatment to offer such patients, they have seldom urged them to return periodically, even for observation, and when they have done so, the patients have seldom come, due to lack of interest. The result is a paucity of follow-up observations on persons who have an "abrupt" type of high-tone loss.

OBSERVATIONS.

The staff of the Otological Research Laboratory at the Johns Hopkins University, aware of the above fact, has in past years encouraged individuals with an "abrupt" type of high-tone loss, whatever the supposed cause, to return from time to time for re-examination, free of any charge or obligation. As elsewhere, not many such patients have actually returned over a period of years. So far as concerns adults, the observations that have been made are in general agreement with the impressions that have also been gained elsewhere (as evidenced in personal conversations of the writer with otologists from this country and from abroad), namely, that impaired hearing of any of the "abrupt" high-tone loss sub-categories remains stationary unless or until there is superimposed on it the effect of some other lesion that causes deafness, a lesion essentially different from the cochlear one supposedly responsible for the high-tone loss present at the first examination, or until additional exposure to severe acoustic trauma causes an extension of the cochlear lesion. The writer hopes in the near future to make a separate report of the observations that have been accumulated with respect to the progression of impaired hearing for high tones in adults; the present report will, except for the above general statements, be limited to a consideration of some interesting observations that have been made with respect to the development and the progression of "abrupt" type high-tone losses in children.

The opportunity to observe the development and the progression of "abrupt" type high-tone losses in children came not as a result of the direct efforts to follow patients with

this type of hearing loss but as a by-product of the departmental program to prevent deafness in children by irradiation of nasopharyngeal lymphoid tissue.[3,4] This program has been carried on in the department for many years, and intensively since 1939. A large number of children have had the irradiation treatment; some of them were participants in a special study made of the hearing of school children,[10] some were patients in the Out-Patient Department of the Johns Hopkins Hospital, and some were private patients of members of the clinical staff of the Sub-Department of Otolaryngology. An essential part of the program has of course been the follow-up examinations. To date, the most comprehensive report of the follow-up study, insofar as the effect of nasopharyngeal irradiation on hearing acuity is concerned, is that made recently by Guild,[9] one item of which has already been mentioned as confirmatory of the idea that the immediate cause of "abrupt" high-tone loss in children is the same as has been proven by histologic observation to be the usual cause in adults.

The present report of this follow-up program concerns children who, while under observation, either developed an "abrupt" type of high-tone loss gradually or had an already existing impairment of this type progress markedly. The great majority of the children with impaired hearing for high tones only have not, when examined several years later, had much progression of the impairments originally present. If marked progressions of impairments for high tones were of common occurrence during the childhood years, the average increase in the high-tone losses for the group of children included in the previous report[9] would have been much greater than actually occurred during the more than six years of observation.

All of the hearing tests included in the present report, like those on which the previous one was based, were made in a sound-proof room at the Otological Research Laboratory, by experienced, well-trained examiners. At each test session, a Western Electric Company 1-A audiometer was used to determine the air-conduction thresholds for tones of 14 frequencies (32, 64, 128, 256, 512, 1,024, 2,048, 2,896, 4,096, 5,793, 8,192,

10,321, 13,004 and 16,384 cycles per second), a 512 d.v. steel tuning fork was used for Weber, Schwabach and Rinne tests, and the hearing acuity for the voice was tested with a W. E. Co. 4-A phonographic audiometer, using the two-digit records. The opposite ear was suitably masked, routinely, during the making of the Schwabach, the Rinne and the voice tests, also, when indicated, during the testing with the pure-tone audiometer. The masking noise was generated in a headphone by a calibrated masking device, locally built,[2] that has been used with satisfaction in the laboratory for many years.

An example of an impairment that did not progress significantly during a nearly 10-year period of observation is included in the case reports given below (see Case 5); comparison of this record with those of the other cases illustrates the difficulty of predicting which children are going to have progression of their impairments during any given period of time.

The four cases of progression or of development of an "abrupt" type high-tone loss reported below include the two most striking examples (see Cases 1 and 3) that have been observed; the selection otherwise has been on the basis of cases that illustrate different forms of progression.

Cases more or less similar to the ones here reported have doubtless been observed by other investigators, but probably, by any one investigator, in such small numbers that publication of the observations did not seem justified. It is hoped that the present report will stimulate others to report the cases they have seen and that from the total assemblage of records better conclusions, especially with respect to causation and to prognosis, can be drawn than are warranted from consideration of the material now available to any one of those who are at present interested in the topic of "abrupt" type high-tone loss. To facilitate the use of these records by others, for the purposes indicated above, the clinical facts about each of the cases here reported are given in more detail than would otherwise seem warranted.

CASE REPORTS.

Case 1: This white boy, J. A., was 10 years old at the time the first examinations were made as part of the study of Baltimore school children.[10] Previously, from the age of 2 years to 5 years, he was seen several times in the Ear, Nose and Throat Dispensary of the Out-Patient Department of the Johns Hopkins Hospital, because of recurring attacks of bilateral suppurative otitis media and cervical adenitis. In 1934, at the age of 5 years, tonsillectomy and adenoidectomy was advised, but the parents refused permission to operate. Three years later, at the age of 8 years, after more episodes of otitis media, the advice to have his tonsils and adenoids surgically removed was accepted and the operation was performed in 1937. Hearing tests were not made at this time.

The physical examination made at the time of the first hearing tests (Dec. 19, 1939) showed that a regrowth of adenoids had occurred; the tubal orifice on the right side was so completely overgrown by lymphoid tissue that it could not be seen through a nasopharyngoscope, on the left side the tubal orifice was partially overgrown by lymphoid tissue. Tonsillar tags were present in both fossae, and the walls of the oropharynx had hyperplastic lymphoid tissue. He reported having had only an average number of colds (2 or 3 a year, each of which lasted about a week) and said he had had no recurrence of otitis media since his operation. Both tympanic membranes were scarred, the left one in the pars flaccida as well as in the anterior-inferior quadrant of the pars tensa, and both were moderately thickened and moderately retracted. Nasal breathing space was good, and the sinuses transilluminated clearly.

The boy said his hearing was good, both ears, as indeed it was except for the tones with frequencies above 4,096 cycles (see audiograms of Dec. 19, 1939, at top of Fig. 1). In the phonographic audiometer test he correctly repeated all the numbers. The sound of the 512 d.v. steel tuning fork was not lateralized from midline positions of the head, this fork was heard better by air conduction than by bone conduction with each ear, and the bone-conduction time for each ear, with the other one suitably masked, was normal for the fork used.[2]

Irradiation of the nasopharynx was advised because of the combination of overgrowth of the tubal orifices by lymphoid tissue, retracted tympanic membranes and impaired hearing for high tones. The advice was accepted and treatments were given, by the use of radon in a brass applicator with walls 1 mm. in thickness, on Jan. 9, Feb. 20, March 27, May 7 and Nov. 14, 1940. All the treatments were given by Dr. Crowe personally.

The examinations made on May 7, before the treatment of this date was given, showed the nasopharyngeal lymphoid tissue to have shrunk until only a moderate amount remained; the left tubal orifice was now open but was somewhat edematous in appearance, and the right tubal orifice was only partially overgrown instead of being completely covered by lymphoid tissue as it had been before treatment was begun. The only other change revealed by the physical examination was that the degree of retraction of the pars tensa of the left tympanic membrane had increased from a rating of moderate to that of marked. In spite of the favorable changes in the nasopharynx, the hearing impairment for high tones had increased, particularly on the left side (compare audiograms of Dec. 19, 1939, and May 7, 1940, in Fig. 1). No change had occurred in his ability to hear the voice (phonographic test) or in the responses to the fork tests.

This boy was next examined after the Summer season, on Oct. 15, 1940, at which time both tubal orifices were normal in appearance, but the right as well as the left tympanic membrane now showed a marked degree of retraction and the hearing impairment for high tones had further increased for both ears (the audiograms for this date are not reproduced in the figure). He was not given another treatment on this date, but when seen a month later he was given his fifth, and final, irradiation treatment, after the hearing tests had revealed a yet further increase in the impairment of thresholds for high tones. By this time the impairment for the left ear, which had been definitely the better in all the previous tests, was about as great as for the right ear (audiograms of Nov. 14, 1940, Fig. 1), which also had suffered a progressive loss of acuity for high tones during the first year of observation.

During the next year this boy was examined on three occasions but was not given further treatment; the lymphoid tissue in his nasopharynx continued to shrink until only a very small amount remained. Both tubal orifices were graded as entirely normal in appearance throughout the year, and the degree of retraction of the tympanic membranes decreased to a rating of moderate. By the end of the year the threshold for the very high frequency of 13,004 cycles had markedly improved in both ears (see audiograms for Nov. 4, 1941, Fig. 1), but the threshold for 8,192 cycles had become worse in both ears, and in the left ear the threshold for 5,793 cycles was poorer than in the right ear, which previously had been much the worse for this tone. The results of the voice and of the fork tests remained unchanged.

The boy was examined again on March 14, 1942, at which time all the findings, including those with respect to hearing acuity, were essentially as they were in November, 1941. He was not seen again until 5 years later, after the war, when he returned, at request, on April 5, 1947. He was then 17 years of age and was working in dusty surroundings, which may account for his statement that he had had many "colds" the past year. Otherwise he had no complaints referable to his nose, throat or ears. The tonsillar tags were still present, but as previously seemed to be causing no trouble. The amount of lymphoid tissue in the oropharynx had decreased greatly, and the nasopharynx was free of lymphoid tissue except for a tiny amount in each fossa of Rosenmueller. Both tubal orifices were normal in appearance. The examiner on this occasion graded the degree of retraction of the right tympanic membrane as moderate and that of the left one as marked. The thresholds for the two ears were now practically identical for high tones (audiograms of April 5, 1947, Fig. 1); the improvement for 13,004 cycles had been lost and the audiograms were typical examples of the "abrupt" high-tone loss type, with 5,793 cycles the lowest frequency involved.

It is interesting to note that during the somewhat more than seven years of observation the threshold for the "border" tone, of the frequency of 5,793 cycles, dropped 35 decibels in the right ear (from the 30 to the 65 db. level) and 55 decibels in the left ear (from the 10 to the 65 db. level.

Comments, especially with respect to causation of the impairment:

The remote cause of the hearing impairment for high tones that developed in this boy is unknown. The immediate cause,

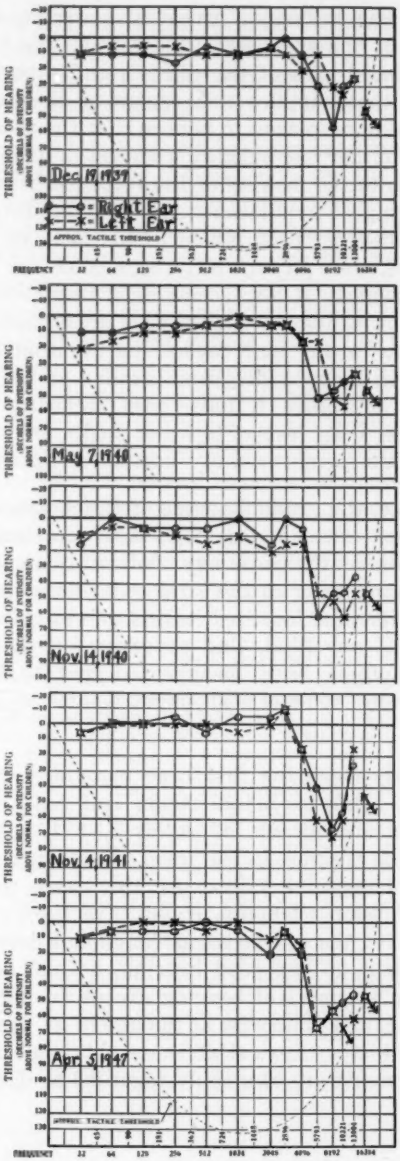

Fig. 1. Five of the 11 pairs of audiograms that were made of a boy who was 10 years old when the first tests of hearing acuity were made and 17 years old when last examined. These audiograms show the progressive increase in the impairment of thresholds for high tones that occurred gradually, and bilaterally, during the period of observation. See text, Case 1.

for each ear, was probably a progressive atrophy of the organ of Corti and of the external sulcus cells in the lower 7 mm. or so of the basal turn of the cochlea, and a partial atrophy of the nerve that supplies this region of the auditory end-organ.[5] There is no reason to believe that the treatments this boy received, either the surgical removal of tonsils and adenoids previous to the making of the recorded hearing tests or the nasopharyngeal irradiations administered during the first year of the observed progression of his hearing impairment for high tones, were in any way responsible for the hearing loss that developed. It also seems most improbable that this boy's recurrent attacks of suppurative otitis media up to the age of 8 years in any way caused his eventual hearing loss for high tones; many audiograms similar to his on April 5, 1947, shown in Figure 1, have been recorded for persons whose histories with respect to otitis media are negative and whose tympanic membranes are normal in appearance.

Furthermore, the gradual course of the development of the functional impairment of this boy's ears practically rules out of consideration as the cause any acute systemic disease, such as mumps or any other of the viral or bacterial infections. It is difficult to imagine localized lesions due to infections, or to toxic agents of any kind, that progress for years after the systemic symptoms have disappeared and that eventually cause bilaterally symmetrical impairments of hearing for high tones.

Had this boy's hearing first been examined when he was 17 years old, after his impairments had reached the stage shown in the audiograms of April 5, 1947 (see Fig. 1), neither acoustic trauma, in the form of exposure to a detonation, nor a congenital failure of the basal turn of both cochleae to develop normally, could have been ruled out as explanations for the symmetrical functional defects present. The boy thought he had good hearing, he had never noticed any change in keenness of hearing except during the attacks of otitis media, and otherwise at the age of 17 years he would have given a history typical of those obtained from persons whose impairments are commonly regarded as due to congeni-

tal cochlear defects or to an entirely forgotten episode of exposure to an explosion. As it is, both of these possible causes of this type of hearing impairment can definitely be eliminated from consideration.

Whether or not this person's hearing impairment for high tones will progress beyond the present stage (as shown in the lowest pair of audiograms in Figure 1) before either normal ageing or other factors deleterious to hearing become superimposed on the existing lesions, is a matter that can be answered only by the passage of time and the making of further careful examinations of his hearing ability. I personally believe that within the next decade of his life the ability to hear the tones for which the thresholds are already severely impaired will be completely lost, but that unless this man is exposed to severe acoustic trauma or has another episode of otitic infection or suffers some less common cause of deafness, he will continue to have normal hearing for speech until presbyacusic changes affect his understanding of consonants. This optimistic opinion is based on the absence, in my experience, of observations in adults of "spontaneous" changes in hearing for high tones comparable to those that occurred during childhood in this boy.

Case 2: This white boy, J. S., was 8 years old when first examined in the Ear, Nose and Throat Dispensary of the Out-Patient Department of the Johns Hopkins Hospital. In spite of a history of frequent attacks of bilateral otitis media and many myringotomies, his tympanic membranes were both normal in appearance except for a marked retraction of the pars flaccida, and the hearing was remarkably good except for the extremely high-pitched tones (see Fig. 2, audiograms of Oct. 10, 1939). He had large tonsils and adenoids, and both tubal orifices appeared to the examiner to be partially occluded by lymphoid tissue. Tonsillectomy and adenoidectomy were performed, at the Johns Hopkins Hospital, Dec. 14, 1939. Hearing tests were not made when he returned a month later for his post-operative check-up, but when he came back 3 months later the audiograms revealed that a marked increase had occurred in the impairment for high tones in the right ear and a slight increase in the left ear (see Fig. 2, audiograms of April 4, 1940). The tonsillar fossae were clean; a moderate amount of lymphoid tissue was seen in the nasopharynx but both tubal orifices were clear. The tympanic membranes now had a moderate degree of retraction of the pars tensa. On April 15, 1940, his nasopharynx was irradiated by Dr. Crowe (2.2 gram-minutes equivalent to each side, using the radon applicator with a brass wall 1 mm. thick). Three weeks later (see audiograms of May 8, 1940, Fig. 2) hearing acuity with the left ear was impaired for all tones, and the "abrupt" type high-tone loss of the right ear had increased slightly for the frequency of 4,096 cycles. The left ear had an 18 per cent loss

for voice (phonographic audiometer test), and the sound of the 512 d.v. steel tuning fork, which had previously not been lateralized from midline points of the head, was referred to the left ear, for which ear the sound was louder by bone conduction than by air conduction and the bone-conduction time was normal. Otoscopic examination of the left ear revealed only a slight hyperemia of Shrapnell's membrane and a moderate retraction of all parts of the tympanic membrane. The boy had a "cold," which had begun about a week previously; no treatment was given this day.

When he returned at the end of the Summer season the hearing of the left ear had improved, but the right ear showed a 40 decibel increase in the previous impairment for 4,096 cycles (audiograms of Sept. 24, 1940, Fig. 2). Both tubal orifices appeared, on nasopharyngoscopic examination, to be partially occluded by lymphoid tissue, and another irradiation treatment was given this day by Dr. Crowe (2.5 gram-minute equivalent, to each side, with the 1 mm. thick brass-walled applicator). A third treatment of 2.44 gram-minutes, with the same applicator, was given Dec. 2, 1940, at which time the hearing was essentially the same as it was in September (audiograms not reproduced). He was examined again in May, 1941, and in October, 1943, but was not given further treatment. He returned, at the request of the Otological Research Laboratory, on April 3, 1947. He reported that he had only occasional mild colds and no sore throats. The tonsillar fossae were clean; a small amount of lymphoid tissue was present in the nasopharynx but both tubal orifices were clearly visualized on nasopharyngoscopic examination. The examiner on this occasion (Dr. Walter Loch) recorded both tympanic membranes as markedly retracted, pars tensa as well as pars flaccida, and as showing definite scarring. The hearing acuity, about 7½ years after the first tests, is shown in the lowest part of Figure 2.

Comment: The threshold for the tone with a frequency of 8,192 cycles was the same in the last test of the left ear as it was in the first test, and for the frequencies adjacent to 8,192 cycles (5,793 and 10,321) in the series tested the thresholds had changed but little. The thresholds for all the other frequencies above 1,024 cycles were, however, definitely poorer for the left ear at the last test than at the first; the greatest change was for 4,096 cycles, at which frequency the audiogram of the left ear has a marked "dip." Whether or not the hearing loss for the left ear of this boy will progress until it reaches the condition present in the right ear is unknown; personally I think the pathologic process in the left ear, whatever it is, had not yet become stabilized when the boy was last examined.

It is of interest to note that for the right ear of this boy essentially all the impairment that developed during the period of observation, except for the "border" frequency of 4,096 cycles, occurred before the nasopharyngeal irradiation treat-

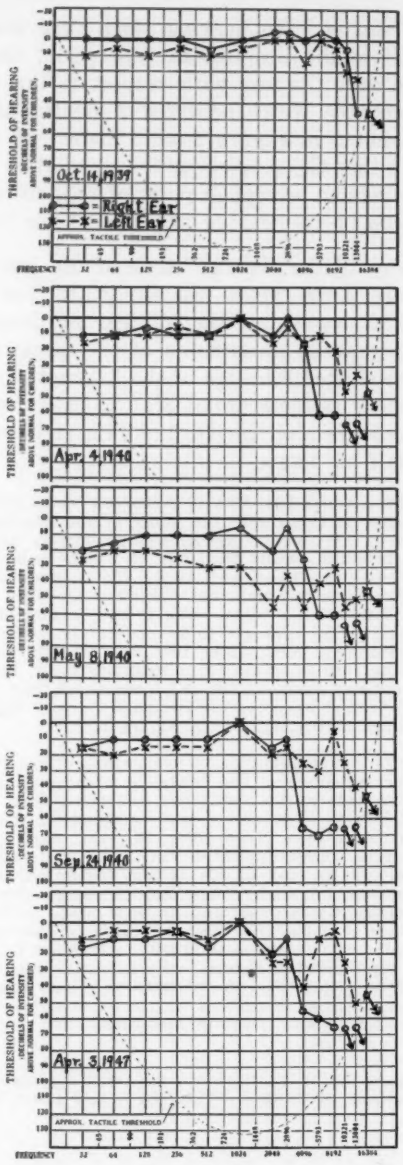

Fig. 2. These 5 pairs of audiograms, of the 8 made, show changes in hearing acuity that occurred during 7½ years in a boy who was 8 years old when the first hearing test was made. See text, Case 2.

ments were started. No hearing tests were made when he was admitted to the hospital for operation, therefore it is impossible to date the loss with respect to the tonsillectomy and adenoidectomy, which occurred 2 months after the first audiograms shown in Figure 2 were made. As with Case 1, I doubt that either the surgical or the irradiative treatment of this boy was related, except in point of time of occurrence, to the progression of his hearing impairment for high tones.

The behavior of the "border" tone of the loss in the right ear of this boy, 4,096 cycles, was similar to that of the "border" tone, 5,793 cycles, of the left ear of Case 1. In both instances the gradual progression of the impairment for the "border" tone shifted the edge of the "abrupt" type high-tone loss first present (audiogram of May 7, 1940, for the left ear of Case 1; audiogram of April 4, 1940, for right ear of Case 2) downward half an octave in pitch.

Case 3: N. B., a white boy, was 12 years old when first examined, on Nov. 9, 1939, as a participant in the study of the hearing of Baltimore school children.[10] The history he gave was entirely negative for ear, nose or throat troubles, except for colds, of which he had about three a year. Nasal breathing space was good; the sinuses transilluminated clearly. The tonsils were small, but the adenoids were large and partially covered both tubal orifices. The tympanic membranes were not thickened or scarred but appeared to be moderately retracted in all parts. With the phonographic audiometer test he correctly repeated all the numbers, and the results of the tests with a 512 d.v. steel tuning fork were likewise entirely normal. The only departures from normal hearing acuity that were found were the impairments for very high tones, shown in the audiograms of Nov. 9, 1939, of Figure 3.

On the basis of the combination of overgrowth of the tubal orifices by lymphoid tissue, retraction of the tympanic membranes and impairment of thresholds for some of the high tones, nasopharyngeal irradiation was advised by Dr. Crowe. The advice was accepted by the boy's parents, and three treatments were given (Nov. 28, 1939; Jan. 4, 1940; April 3, 1940) with radon in a brass applicator that had a wall thickness of 1 mm. The tests made on the day of his second treatment showed that his hearing impairment for high tones had increased considerably; this increased impairment was confirmed by tests made in February and in April. The stages of impairment that had been reached by May 6, 1940, a month after his last irradiation treatment, are shown in the second pair of audiograms reproduced in Figure 3. By this time only a small amount of lymphoid tissue remained in the nasopharynx, both tubal orifices were normal in appearance on inspection with a nasopharyngoscope, and the degree of retraction of both tympanic membranes had receded to a rating of slight.

The appearance of the nasopharynx and of the tympanic membranes remained essentially unchanged during the next 8 years, but the hearing

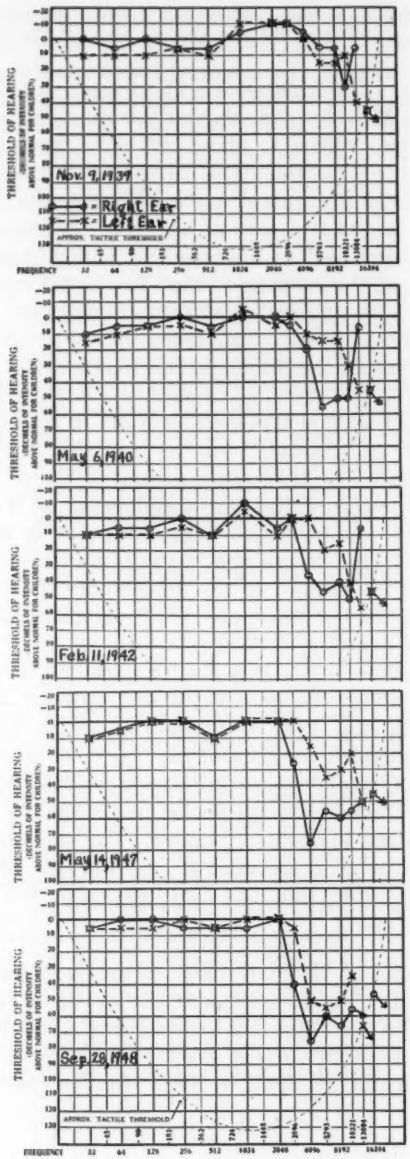

Fig. 3. Five of the 12 pairs of audiograms that were made during a nearly 9-year period of observation of a boy who was 12 years old when first examined. The marked degree of bilaterally impaired hearing for high tones developed gradually. See text, Case 3.

impairments for high tones progressed, in the manner shown in the audiograms reproduced in the lower three parts of Figure 3.

Comments: The 5 pairs of audiograms reproduced in Figure 3, of the 12 tests made during the nearly nine-year period of observation, suffice to show not only the amount of the increase in the impairments of thresholds for high tones that occurred bilaterally but also the difference in the order of progression in the two ears of this boy. In the right ear the progression occurred as a deepening and widening, towards lower tones, of the moderate "dip" for 10,321 cycles that was present when he was first examined (audiogram for right ear on Nov. 9, 1939). Only after more than 2 years of this type of progression did the threshold for 13,004 cycles, at the high-frequency side of the "dip," begin to be impaired. During its drop from the normal threshold of 5 db. to that of 60 db., the threshold for the "border" tone at the other side of the widened "dip" also dropped from a normal of "zero" to the 40 db. level (2,896 cycles, right ear, audiograms of Feb. 11, 1942, May 14, 1947, and Sept. 28, 1948). He was in the Army for two years shortly before the re-examination in 1947, which was made at the request of the laboratory; the only exposure to loud noise during this time that he could recall was a session of rifle practice, following which his ears "rang for a week." Whether or not this acoustic trauma was responsible for part or all of the increase in impairment that occurred between 1942 and 1947 is unknown, but it is known that further impairment for both of the "border" tones (2,896 and 13,004 cycles) occurred during the next year after the test made in 1947, during which period he worked in an office and was not exposed to acoustic trauma. The last audiogram, for the right ear, is a typical example of "abrupt" high-tone loss, of the sub-category that has 2,048 cycles as the highest frequency well heard.[7]

The left ear of this boy had, at the first examination, a slightly greater average impairment of thresholds for high tones than did the right ear (audiograms of Nov. 9, 1939, in Fig. 3). This situation was reversed by the time the second tests were made, on Jan. 4, 1940 (audiograms not repro-

duced), and from that time on the impairment of the right ear for high tones was the greater. The progression of the impairment of the left ear occurred somewhat irregularly from the high-frequency end of the scale towards tones of lower frequencies, not by a deepening and widening of a clearly recognized "dip," as in the case of the right ear. At the last examination (see audiogram of Sept. 28, 1948) the impairment for high tones in the left ear was clearly of the "abrupt" type, but the highest frequency well heard was a half-octave higher in pitch than in the right ear.

Two brothers of N. B. also participated in the study of the hearing of Baltimore school children.[10] They were aged 10 and 8 years, respectively, when first examined in the Autumn of 1939, and the younger one, like N. B., had slight impairments of threshold for some of the very high-pitched tones. Like the older brother, the histories of these boys were entirely negative with respect to ear, nose or throat troubles or treatments. None of the three had had tonsils or adenoids surgically removed. Both of the younger brothers had more lymphoid overgrowth of the nasopharyngeal orifices of the Eustachian tubes than did the older brother who was treated with radon, but irradiation was not advised for either of them because their tympanic membranes were rated as only slightly retracted. Because of the large adenoids, however, both brothers were re-examined twice yearly until the study had to be discontinued on account of a shortage of staff after the United States entered World War II. During the period of re-examination (until October, 1941, for one boy and January, 1942, for the other) hearing acuity and conditions of tympanic membranes and of nasopharyngeal lymphoid tissue remained essentially unchanged. Both of these untreated brothers were re-examined in May, 1946, at which time the hearing thresholds of both were essentially the same as they had been in 1939. The tubal orifices of one of them, the younger, were still partially overgrown by lymphoid tissue; in the other boy the nasopharyngeal lymphoid tissue had spontaneously regressed until only a small amount remained and the tubal orifices were both normal in appearance.

The above observations should not be interpreted to mean that the irradiation received by the older brother (see Case 3) caused the hearing impairment for high tones that he developed; *i.e.*, that if he had been handled as were the younger brothers, his marked degree of impaired hearing for high tones would not have occurred. As has been stated in the comments on Cases 1 and 2, I do not believe the real cause of such impairments is in any way affected by either surgical or irradiative therapy. In my opinion, it was in each case merely a coincidence that the treatments were given during the period when the causative lesions, whatever they are, were in the progressive stage. The fact that the hearing impairments of the untreated brothers did not progress, while those of the treated brother did increase markedly, illustrates well, however, the difficulties of prognosis in individual cases.[8]

Case 4: B. M., a white boy, was 13 years old when first examined as a participant in the study of the hearing of Baltimore school children.[10] At the age of 9 years he had had a tonsillectomy and adenoidectomy; otherwise his ear, nose and throat history was negative except for about 2 mild colds per year. Both tympanic membranes were normal in appearance in spite of the fact that the adenoids were large and partially covered the nasopharyngeal orifices of both Eustachian tubes. He heard correctly all the numbers in the phonographic audiometer test, and the tests with a 512 d.v. steel tuning fork also yielded normal data for both ears. With the pure-tone audiometer the thresholds of the right ear for high tones were near the lower limit of the "normal" range. The tests of the left ear revealed a "dip" to the 50 db. level for 5,793 cycles, and some impairment for 8,192, 10,321 and 13,004 cycles; the threshold for the highest tone, 16,384 cycles, was in the normal range for this frequency (see audiograms of Dec. 7, 1939, in Fig. 4).

A nasopharyngeal irradiation treatment, with radon in a brass applicator with a wall thickness of 1 mm., was given by Dr. Crowe on Dec. 18, 1939. When the boy returned five weeks later the adenoids were not so large as previously and both tubal orifices were normal in appearance, but the hearing thresholds for high tones had become worse in both ears (audiograms of Jan. 23, 1940, Fig. 4). His temperature was slightly elevated, but he had no other signs of a "cold." He was not treated this day and was not re-examined again until June, 1940, at which time the hearing acuity of both ears had improved slightly (audiograms not reproduced) and the tubal orifices were still normal in appearance. He was next seen on Dec. 17, 1940, at which time the thresholds for the right ear were as good as at the first test, but the impairment of the left ear had progressed. The "dip" at the frequency of 5,793 cycles, present at the first examination, had become wider in both directions; the threshold for 4,096 cycles had dropped from the 15 db. to the 45 db. level, and the impairments of the thresholds for all the higher frequencies, except 13,004 cycles, had also increased (see audiograms of Dec. 17, 1940, Fig. 4). The tubal orifices were normal in appearance, as they had been since the examination in January, 1940, but the tympanic membranes were now

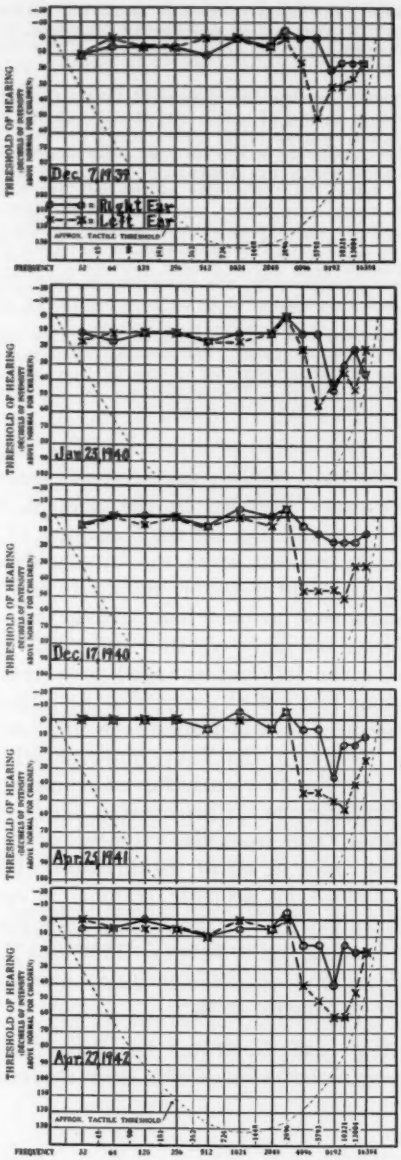

Fig. 4. The changes in hearing acuity for high tones that occurred during 2 years and 4 months in a boy 13 years of age when first examined are shown by the 5 pairs of audiograms of the 9 made, that are reproduced. Note the good hearing, in this case, for the highest frequency tested, 16,384 cycles per second. See text, Case 4.

rated as moderately retracted instead of as normal. The adenoids were rated as moderate in size and as not having regressed beyond the stage observed in June, so a second radon treatment of the nasopharynx was given Dec. 17, 1940.

When examined 2 months later, Feb. 25, 1941, the adenoids had decreased in size to a rating of small, but the thresholds of the right ear for the four highest tones (8,192 cycles upwards) had dropped to the 30 db. level; the thresholds of the left ear were essentially the same as in December (audiograms not reproduced). At the next examination, made 2 months later, the thresholds of the left ear had changed only slightly, but the thresholds of the right ear for the three highest tones had improved and its audiogram showed a "dip" to the 35 db. level for the frequency of 8,192 cycles (audiograms of April 25, 1941, in Fig. 4). The physical examination on this date was made by Dr. Crowe, who rated the tympanic membranes, the nasopharynx, including the fossae of Rosenmueller, and the tubal orifices as now normal in appearance.

During the next year this boy was re-examined three times (November, 1941; January, 1942; April, 1942). The hearing impairments for the tones from 4,096 cycles upwards persisted in the left ear, as did the "dip" at 8,192 cycles in the right ear. The audiograms of the last test, made on April 27, 1942, are reproduced in Figure 4.

Comments: The period of observation of this boy, B. M., was only a little more than 2 years, a much shorter time than for the other cases included in the present report. It is unusual to have the hearing for the frequency of 16,384 cycles remain so nearly normal when the thresholds for all other frequencies above 2,896 cycles are impaired, and it would have been of much interest to have had further tests of this boy's hearing, to learn not only whether the "dip" nature of the impairment for the left ear changed to a typical "abrupt" type of high-tone loss but also whether the impairment of the right ear progressed by widening of the "dip" at 8,192 cycles, or otherwise. He was in the Navy, however, when the laboratory tried to get him to return for re-examination in April, 1947, and could not come. If his hearing is carefully tested, at some place other than Hopkins, while he is in the Navy or after discharge from it, all of the high-tone loss found will probably be blamed on acoustic trauma, a conclusion that would not be supported by the changes known to have occurred in his hearing acuity between 1939 and 1942. The case illustrates nicely one of the difficulties encountered in the interpretation of hearing tests.[8]

The following case illustrates the usual experience with respect to progression of a slight or a moderate degree of

impaired hearing for high tones in childhood, namely, that the thresholds fluctuate a bit but do not change greatly over a period of years. When the first hearing tests of this boy are compared with those of Cases 1 to 4, the case also illustrates the difficulty of predicting, on the basis of the early examinations, whether or not a patient's hearing impairment for high tones is going to be progressive in spite of treatment.

Case 5: This boy, J. M., a private patient of Dr. Crowe, was 9 years old when first examined, Oct. 16, 1939. His audiograms for that date are reproduced at the top of Figure 5. With the phonographic audiometer test he heard the two-digit numbers normally with the right ear but had a 6% loss with the left ear. The sound of the 512 d.v. steel tuning fork was not lateralized from midline points of the head, and the fork was heard for normal times by air conduction and by bone conduction with both ears.

The boy had had several attacks of otitis media between the ages of 2 and 7 years, but none since his adenoids were removed for the second time, in May, 1937. He had had a tonsillectomy and adenoidectomy a few months previously. Nasopharyngoscope examination in October, 1939, revealed that he again had a marked degree of overgrowth of both tubal orifices by hyperplastic lymphoid tissue. Both tympanic membranes were markedly retracted in all parts, and the left one was atrophic and had a healed perforation posteriorly.

A week later, on Oct. 23, 1939, his nasopharynx was irradiated with radon in a brass applicator with a wall thickness of 1 mm.; the dosage given to each side was 1.9 gram-minutes equivalent. The treatment was repeated on Nov. 29, with a dosage of 2.29 gram-minutes equivalent to each side. The audiograms made a month later (Dec. 29, 1939) are reproduced in Figure 5. Comparison of the thresholds of the right ear on Oct. 16 and Dec. 29 shows that a considerable drop had occurred in the hearing acuity for high tones, and gives the impression, not borne out by the later tests, that one is dealing here with a condition like that found in the previous cases (Nos. 1 to 4). The nasopharyngeal lymphoid tissue had decreased markedly in amount but the tubal orifices were not quite normal in appearance. The appearance of the tympanic membranes had not changed. He was not treated on this occasion but was given another irradiation treatment, his third, when he returned on March 1, 1940. The audiograms of that date (not reproduced) showed less hearing loss, for the right ear, than in December, but more than at the time of the first test. The left ear had slightly more impairment for high tones than it had in December, but its hearing acuity for low and for middle range tones had improved.

The boy was examined again in June, 1940, at which time, in spite of having but recently had whooping cough, the nasopharyngeal lymphoid tissue had entirely disappeared and the tubal orifices, for the first time, were normal in appearance. The tympanic membranes were as at previous examinations. The hearing (audiograms not reproduced) of the left ear had improved slightly for high tones. When next seen, five months later, the thresholds of the left ear for high tones had again improved slightly and the thresholds of the right ear were in the normal range (see audiograms of Nov. 6, 1940, Fig. 5), but there was some lymphoid tissue present in both fossae of Rosenmueller and the posterior lip of

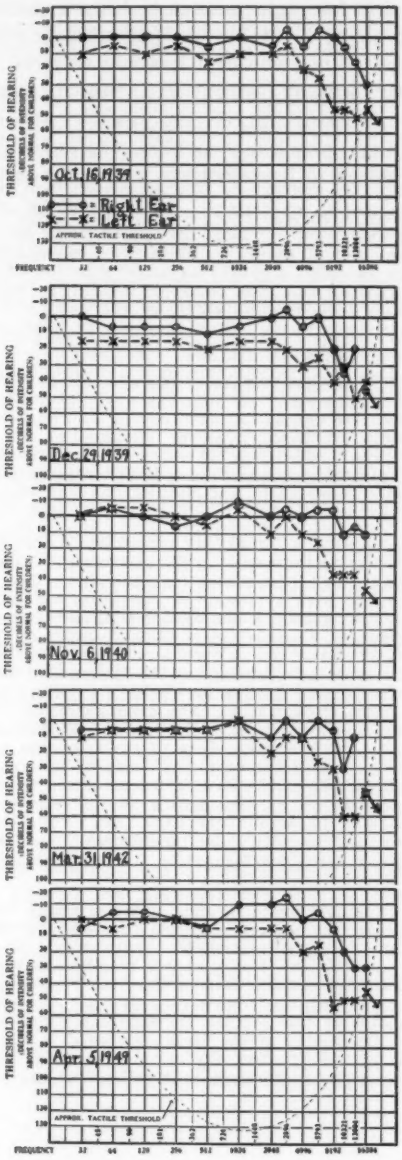

Fig. 5. This series of 5 pairs of audiograms, of the 13 made during a nearly 10-year period of observation of a boy who was 9 years old when first examined, illustrate the usual experience of children who have slight to moderate impairments of thresholds of hearing for high tones, namely, fluctuations of the thresholds but only a very little permanent progression of the impairment See text, Case 5.

the right Eustachian tube orifice was red and granular in appearance. He was given his fourth irradiation treatment this day, and a fifth treatment was given on Feb. 17, 1941, at which time the orifice of the right tube was still red and granular and the hearing acuity for this ear for very high tones had dropped slightly (audiograms of this date not reproduced). The lymphoid tissue on the lateral walls of the pharynx, particularly on the right side, which had been noted at the earlier examinations, had increased in amount, and it also was given an irradiation treatment on Feb. 17, 1941.

When the boy returned nearly a year later (Jan. 28, 1942), the hearing acuity of the left ear for high tones was worse than in any of the audiograms reproduced in Figure 5, the orifice of the right Eustachian tube was still not normal in appearance and there was much hypertrophied lymphoid tissue on the lateral walls of the pharynx, particularly on the right side. Thresholds for the right ear had dropped only slightly; the readings for the three highest frequencies were 25, 20 and 35 decibels, respectively. An irradiation treatment was given to the right side of the nasopharynx and to the right side of the pharynx.

Two months later, when next seen, the pharynx and the nasopharyngeal orifice of the right tube were so normal in appearance that he was not given another treatment. The audiograms made at this time are reproduced in Figure 5 (audiograms of March 31, 1942). He was not seen again until Feb. 5, 1943, at which time the nasopharynx and the pharynx were still normal in appearance, but he complained of a cough which he said had bothered him since he had whooping cough in the Spring of 1940. No cause for the cough was found. Special hearing tests were not made on this occasion, but when he was seen in October, 1943, the thresholds for high tones were found to be better, for both ears, than they had been in March, 1942 (see Fig. 5). There was, however, a small amount of lymphoid tissue present in both fossae of Rosenmueller and he was given another irradiation treatment, his last (as of the date of writing), to both sides of the nasopharynx and to both lateral walls of the pharynx. The dosages were: 2.3 gram-minutes equivalent, with radon in the 1 mm. thick brass-walled applicator, to each side of the nasopharynx, and 1.9 gram-minutes equivelent to each lateral wall of the pharynx. At this time he had had no ear infection since more than two years before his first irradiation treatment, and for the past three years he had had only about one cold per year, each of which was of brief duration.

When he was next seen, in June, 1945, the boy reported having had no colds the previous winter; his nasopharynx and pharynx were in good condition and his hearing was better than it had been at any previous examination. Even for the left ear the only impairments of thresholds were for the three frequencies above 8,192 cycles. In September, 1947, the physical conditions and the hearing thresholds were about the same as in 1945. By April 5, 1949 (see audiograms of that date, Fig. 5) the hearing thresholds for high tones had again dropped to levels that average slightly worse than he had at the beginning of the period of observation, nearly ten years previously. He complained now of a chronic post-nasal discharge, which he said was of several years' duration. He was found to be sensitive to dust, cats, and feathers. No evidence of sinus infection was found and the nasopharynx and the pharynx were both entirely free of lymphoid tissue. The tympanic membranes had not changed materially in appearance since the first examination of them was made.

DISCUSSION AND CONCLUSIONS.

The facts presented above show that the "abrupt" type of high-tone loss can, and sometimes does, develop or progress markedly during childhood, and that the development or the progression of this type of impaired hearing may occur gradually, over a period of years.

The evidence further shows that for the cases reported neither of the usual diagnoses with respect to the remote or true cause of this type of hearing impairment would be correct. A history of exposure to acoustic trauma of the blast type could be elicited in only one of the four cases, and in this instance the exposure occurred after a major portion of the boy's hearing impairment had developed. The other of the two usual diagnoses, namely, congenital cochlear lesion, malformation or failure to develop, obviously would be incorrect for any of the cases cited, except in the sense that the diagnosis might imply inheritance of a tendency to develop a cochlear lesion of the basal turn long after birth. This interpretation of the term congenital naturally cannot, on the basis of the available evidence, be ruled out of consideration as the possible remote cause of the impaired hearing that occurred in these children while they were under observation.

Either to prove or to disprove that the tendency to develop this type of hearing impairment during childhood is inherited, it would be necessary to have available, for the corresponding years of their lives, similarly detailed data with respect to the hearing acuity of the relatives of the children who have participated in the follow-up studies reported. Observations with respect to the present status of the hearing of the parents and of the other adult relatives of the children would not suffice to decide this question. In this connection, it should be recalled that Ciocco's data,[1] cited in the opening paragraph of this paper, indicate that about 60 per cent of adults have some form or other of this type of hearing impairment.

The high incidence of "abrupt" high-tone losses in adults, and especially the great difference in the incidence of this type of hearing impairment in children and in adults (see

above), makes it seem probable that a follow-up study of the hearing acuity of a large number of persons, covering the age period from childhood to middle age, would reveal numerous examples like those reported in this paper. It seems improbable, in view of the evidence now available, that exposure to acoustic trauma of the blast type accounts, together with possible congenital lesions, for the high incidence of "abrupt" type high-tone losses in adults. A follow-up study such as suggested would of necessity take many years to complete, and would require the long-time cooperation of many persons. Such a follow-up study would, however, doubtless yield basic information about the causation of the most common form of impaired hearing that occurs in man. The study would also afford a better basis than now exists for clinical prognosis and for advice to patients who have this type of hearing impairment. To make such a study of a significantly large sample of the population, making allowances for the inevitable disappearance of many of the cooperating subjects, would require financial support of considerable magnitude.

As was stated in the comments on Case 1, the evidence available with respect to the examples reported does not support the idea that infection, either bacterial or viral, is commonly the remote or true cause of the "abrupt" type of high-tone loss. Such a hypothetical cause is not completely ruled out of consideration by the available evidence, however, primarily because the number of cases that have been observed during progression of the impairment is small. The crucial evidence needed to determine this point, as well as others, could be obtained by analysis of the observations that can be made in a lengthy follow-up study such as indicated in the preceding paragraph.

The development or the progression of "abrupt" type high-tone losses is not causally related to nasopharyngeal lymphoid tissue near the tubal orifice, or elsewhere, so far as the observations of the present paper indicate. In each of the cases reported shrinkage of the hyperplastic lymphoid tissue present at the beginning of the period of observation was successfully and promptly accomplished by means of irradiation

with a radon applicator. Progression of the impairment of hearing for high tones continued, however.

That the treatment itself caused the development or the progression of the impairment seems extremely improbable, in view of the known high incidence of this type of impaired hearing in adults before the days of nasopharyngeal irradiation, and in view of the additional fact that most of the children who have had the irradiation treatment have not, during a period of time similar to that of the present follow-up study, developed or had a marked progression of an "abrupt" type of high-tone loss.[9] Any causal relationship that may appear, from perusal of the case reports of the present paper alone, to exist between nasopharyngeal irradiation and "abrupt" type high-tone loss should be interpreted in the light of the circumstance that the follow-up studies of children have been associated with this method of treatment. In other words, the superficially apparent relationship is most probably nothing more than a temporal coincidence; treatment was given during the age period when the hearing impairment was developing, or progressing, for some other reason, which is as yet unknown, or unrecognized.

SUMMARY.

The "abrupt" type of high-tone loss is discussed from the standpoint, primarily, of remote or true causes of the lesion of the basal turn of the cochlea usually regarded as the immediate cause of the functional impairment.

Four cases are reported, in considerable detail, of children who while under observation either developed an "abrupt" type of high-tone loss or had a marked degree of progression of an already existing impairment of this type. In all four instances, the impairments progressed gradually.

The cause of the impairments, had the last examinations of these children been their first, would have been regarded by most otologists as either an exposure to acoustic trauma of the blast type or a congenital cochlear lesion. The reported observations show that neither diagnosis would be correct for any of these four cases.

Other hypothetical, or even plausible, causes are discussed in the light of the evidence from the reported cases. All are ruled out of consideration, more or less completely, and the conclusion is reached that the remote or true cause of many instances of "abrupt" type high-tone loss is as yet unknown, or unrecognized.

The suggestion is made that a follow-up study of a large number of persons from childhood until they reach mid-adult age would provide basic information about the most common type of impaired hearing that occurs in man, and that such a study would also furnish a better basis than now exists for clinical prognosis and advice.

REFERENCES.

1. Ciocco, A.: Observations on Hearing of 1,980 Individuals: A Biometric Study. The Laryngoscope, 42:837-856, Nov., 1932.

2. Committee Report, American Otological Society: Methods of Testing Hearing by Bone Conduction. *Ann. Otol., Rhinol. and Laryngol.*, 45:801-823, Sept., 1936.

3. Crowe, S. J.: Irradiation of the Nasopharynx. *Tran. Amer. Acad. Ophthal. and Otolaryngol.*, pp. 29-35, Sept.-Oct., 1946.

4. Crowe, S. J., and Baylor, J. W.: The Prevention of Deafness. *Jour. A. M. A.*, 112:585-590, Feb. 18, 1939.

5. Crowe, S. J.; Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High-Tone Deafness. *Bull. Johns Hopkins Hosp.*, 54:315-379, May, 1934.

6. Guild, S. R.: War Deafness and Its Prevention—Report of the Labyrinths of the Animals Used in Testing of Preventive Measures. *Jour. Lab. and Clin. Med.*, 4:153-180, Jan., 1919.

7. Guild, S. R.: A Method of Classifying Audiograms. The Laryngoscope, 42:821-836, Nov., 1932.

8. Guild, S. R.: Interpretation of Hearing Tests. *Arch. Otolaryngol.*, 49:431-437, Apr., 1949.

9. Guild, S. R.: Nasopharyngeal Irradiation and Hearing Acuity: A Follow-Up Study of Children. The Laryngoscope, 60:55-76, Jan., 1950.

10. Guild, S. R.; Polvogt, L. M.; Sandstead, H. R.; Loch, W. E.; Langer, E.; Robbins, M. H., and Parr, W. A.: Impaired Hearing in School Children. The Laryngoscope, 50:731-746, Aug., 1940.

11. Hoessli, H.: Weitere experimentelle Studien über die akustische Schädigung des Säugetierlabyrinths. *Ztschr. f. Ohrenheilk.*, 64:101-145, 1912.

12. Wittmaack, K.: Zur Frage der Schädigung des Gehörsorgans durch Schalleinwirkung. *Ztschr. f. Ohrenheilk.*, 59:211-220, 1909.

13. Yoshii, U.: Experimentelle Untersuchungen über die Schädigung des Gehörsorgan durch Schalleinwirkung. *Ztschr. f. Ohrenheilk.*, 58:201-251, 1909.

ON THE CHEMICAL COMPOSITION OF THE HUMAN PERILYMPH AND ENDOLYMPH.*†

JULES G. WALTNER, M.D., and SAMUEL RAYMOND, Ph.D.,
New York, N. Y.

Many open questions are still awaiting an answer in the basic physiology of the human inner ear. Knowledge of the chemical nature, source and place of resorption of the labyrinthine fluids is essential before any understanding of the normal or pathological physiology of the inner ear can be expected. But little progress is possible in research on the etiology of otosclerosis, streptomycin lesions, certain types of deafness such as Ménière's, etc., without this knowledge. Our therapeutic efforts in the same diseases are therefore haphazard or at best symptomatic.

No data are known on the chemical structure of the labyrinthine fluids in man. The commonly accepted hypothesis that the origin of the perilymph is from the spinal fluid through a communication between the subarachnoidal space and the scala tympani of the basal coil of the cochlea via the cochlear aqueducts lacks proof. Under this hypothesis a normal flow of cerebrospinal fluid should take place in the cochlear aqueduct from the meninges to the scala tympani. Perilymph, therefore, should be of identical nature with cerebrospinal fluid.

Previous histological studies of one of the authors[1] on both human and animal temporal bones in serial sections pointed to the existence of a membranous structure at the cochlear ending of the cochlear aqueduct which seemed to act as a barrier membrane separating perilymph from spinal fluid. Studies on human embryos seemed to confirm this finding, which was also

*From the Department of Otolaryngology and Bacteriology, College of Physicians and Surgeons, Columbia University, and the Presbyterian Hospital, New York, N. Y.

†This study was assisted by grants from the Research Council of the American Otological Society and the U. S. Public Health Service.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 12, 1950.

sustained by findings in temporal bones with purulent meningitis. Animal experiments were performed to study perilymph-spinal fluid relationship with the Prussian blue and chicken blood methods which equally made a free flow of spinal fluid into the cochlea questionable.[2]

The inaccessible anatomical location of the human labyrinth and possible trauma with destruction of hearing and vestibular function made it impossible to obtain perilymph or endolymph previously. Surgical procedures like Day's operation allowed us to obtain samples of perilymph from living human subjects. After the window of the lateral semicircular canal was completed the field was cleaned of blood and made dry. With the help of a loupe a glass capillary was then introduced into the perilymphatic space and a very gentle suction was applied, obtaining a small sample of the fluid. Immediately following operation spinal fluid was obtained by a spinal tap. Five specimens of perilymph were thus obtained. One specimen of endolymph was collected after tearing the endolymphatic duct during a Portmann operation. All six patients were operated upon for Ménière's disease.

METHOD.

The samples from each patient as obtained for analysis consisted of *1.* a 3 to 44 mg. sample of ear fluid, *2.* a sample of spinal fluid, and *3.* a sample of blood which had been diluted 1:100 in a hemocytometer. The ear sample was contaminated with small amounts of blood. This fact, together with the small quantity available, limited the possibilities for analysis and the accuracy obtainable. Two types of measurement were made: ultraviolet absorption and total nitrogen. The ultraviolet measurements were particularly useful since they allowed the amount of blood contamination of the samples to be calculated from the hemoglobin peak at 410.

The spectrum of ear fluid showed three features: a peak at 410, another at 275, and a sharp end absorption beginning at 265 millimicrons (see Table I). The peak at 410 was due to hemoglobin from the blood contamination. By measuring the

TABLE I.

ULTRAVIOLET ABSORPTION OF PERILYMPH FROM DAY OPERATION.

Specimen	Conc. %	D (275)	D (410)	Blood $\frac{D275}{D410}$	Blood Correction (Optical Density)	Corrected E (1% 1 Cm.)
1	2.07	.394	.810	.332	.269	0.060
2	2.68	.632	.665	.513	.340	0.109
3	25.5	.742	.336	.398	.118	0.024
4	15.2	.800	.880	.542	.477	0.021
5	13.8	.393	.298	.410	.122	0.020
6*	1.35	.128	.175	.481	.084	0.052
	2.46	.250	.353	.481	.170	0.054

*Endolymph.

hemoglobin absorption of the corresponding blood sample and comparing it with the corresponding absorption of the ear fluid, the quantitative measure of the amount of contamination was obtained. The peak at 275 was probably due to the tryptophanetyrosine content of the protein present, although a number of other substances such as purines and ascorbic acid absorb appreciably at this wave length and were probably present in the fluid.

The spectrum of spinal fluid showed none of the features of ear fluid. The 410 absorption was very low or absent because this fluid was obtained blood-free. The absorption at 275 did not show a peak, but only a slight shoulder in the rise of the curve to the end absorption at 265. It is evident from the analysis of the two sets of spectra that there are marked quantitative and qualitative differences in the absorbing substances present in the two fluids.

TABLE II.

ULTRAVIOLET ABSORPTION OF SPINAL FLUID.

Specimen	Conc. %	D (275)	D (410)	Blood $\frac{D275}{D410}$	Blood Correction (Optical Density)	Corrected E (1% 1 Cm.)
1	8.40	.092	.005	.332	.001	0.008
2	42.4	.605	.030	.513	.015	0.014
3	100	1.010	.026	.398	.010	0.010
4	100	.990	.277	.542	.150	0.008
5	100	.930	.025	.410	.010	0.009
6	15.7	.177	.077	.481	.037	0.009

The ultraviolet absorption of the fluids is given in Table I and II, together with the extinction coefficients E (1 per cent 1 cm.) calculated from them. Correction was made for the blood contamination.

After ultraviolet measurements were made the total sample of ear and spinal fluids was quantitatively transferred to digestion flasks and digested with Kjeldahl mixture. Because of the large blood dilution (1:1000 or 1:2000), 1 ml. of the blood dilution was digested. The ammonia in the digest was determined by the microdiffusion method of Conway. Correction was made for blood N in the fluids (see Table III).

TABLE III.

TOTAL N.

Perilymph.

Specimen	Weight (Mg.)	Total N Microgram	Blood N N/D 410	Corrected N Mg. %	Blood Correction Microgram
3	44	13	15	28	0.9
4	20	8.1	22	27	2.6
5	18	5.1	17	25	0.6

Spinal Fluid.

3	133	17	15	13	0.0
4	157	22	22	13	0.9
5	110	16	17	14	0.2

DISCUSSION.

The small number of cases, the minute amounts of inner ear fluids, and the unavoidable contamination of the fluids with microscopic amounts of blood make it necessary to consider the results as not final.

The result of these measurements, however, allow us to draw a number of conclusions. The protein content of perilymph and endolymph as measured by D-275 was more than double that of the spinal fluid in each specimen. Measurements of total N confirmed results obtained with the ultraviolet absorption, thus giving a double check on the results.

The huge difference between the protein contents of the two fluid systems disproves our previous concepts of identity of perilymph and cerebrospinal fluid. In addition, a free flow of spinal fluid into the cochlea does not appear possible in view of the definite difference in chemical structure of the two fluid systems. Measurements of Na content were obtained in one of these cases and showed it is present in almost identical concentration in both spinal fluid and perilymph.

The results give additional support to the existence of a barrier membrane[1] at the cochlear opening of the aqueduct. This membrane apparently does not permit passage of large molecules of colloids like proteins. Equal concentration of Na on the two sides of this membrane, on the other hand, is strongly suggestive of a semipermeable nature of the barrier membrane.

Experiments performed by Gisselsson[3] and Altmann and Waltner[2] showed that crystaloids diffuse through this membrane in monkeys, cats and rabbits.

Perilymph probably is derived from at least two sources. One of these is the cerebrospinal fluid which supplies some of the crystalloids like Na, etc., which are able to diffuse through the membranous barrier. A similar diffusion of crystalloids equally may take place through Reissner's membrane from the endolymph into the perilymph. Experimental evidence exists in cats and rabbits showing an easy passage of iron salts through intact Reissner's membrane.[2] Identification of the source of proteins and other large molecular components of the perilymph still awaits additional evidence.

Protein content of the endolymph studied in one single case of Ménière's disease revealed that it is in the same range as the perilymph. This single specimen, although two measurements gave identical results, would have little significance alone except that it fits very well into the picture pieced together with the help of animal experiments (cats) by Aldred, Hallpike and Ledoux[4,5] which demonstrated the almost identical osmotic pressure and index of refraction of the endo- and perilymph.

A large series of experiments on cats were performed by us, using the same method of measurement as on human material. They allow us to make the same conclusions on the protein content of the perilymph. These studies will be published in a separate paper.

It still has to be seen whether these conclusions can be applied without change to the normal perilymph, or are valid only for perilymph and endolymph in Ménière's disease.

With perfection of the method of collecting samples of perilymph it may become possible to obtain perilymph from a larger number of suitable cases of otosclerosis during fenestration operation without impairing the function of the ear.

SUMMARY.

1. Five specimens of perilymph were obtained from the lateral semicircular canal of humans while performing Day operations for Ménière's disease.

2. One specimen of endolymph was collected from a patient with Ménière's disease during a Portmann operation.

3. Measurements of the ultraviolet absorption and total nitrogen were made on these fluids.

4. The protein content of the inner ear fluids as indicated by the above measurements was more than twice that of the cerebrospinal fluid of the same patients.

5. These figures corroborate the existence of a barrier between perilymph and spinal fluid, probably of semipermeable nature.

Thanks are expressed to Mr. Herbert Wohl and Mr. Robert Franzl for technical assistance.

REFERENCES.

1. WALTNER, J. G.: Barrier Membrane of the Cochlear Aqueduct. *Arch. Otolaryngol.*, 39:656-669, 1948.

2. ALTMANN, F., and WALTNER, J. G.: The Circulation of the Labyrinthine Fluids. *Ann. Otol. Rhinol. and Laryngol.*, 56:684, 1947.

3. GISSELSSON, L.: Passage of Fluorescein Sodium to Labyrinthine Fluids. *Acta Otolaryngol.*, 37:268, June, 1949.

4. ALDRED, P.; HALLPIKE, C. S., and LEDOUX, A.: Observations on the Osmotic Pressure of the Endolymph. *Jour. Physiol.*, 48:446, 1940.

5. LEDOUX, A.: Indice de Refraction des Liquides Cephalorachidien et Labyrinthiques du Chat. *Acta Biol. Belg.*, 1:506, 1941.

180 Ft. Washington Avenue.
630 W. 168th Street.

AN EXPLANATION OF CERTAIN TYPES OF TINNITUS AND DEAFNESS.*

(Lantern Demonstration.)

EDMUND PRINCE FOWLER, M.D., and
EDMUND PRINCE FOWLER, JR., M.D.,
New York, N. Y.

During the last one hundred years the advances in medicine have brought us to a stage where we now can examine properly prepared tissue under high magnification, and see microscopic morphological changes resulting from disease. We also have learned much about the infectious diseases and something about why they cause disturbances of the tissues and many ways in which they may be combated and cured. We know that mechanical and acoustic traumas and poisons as well as certain diseases may and frequently do cause tinnitus and deafness. We know something about the inheritance of deafness. We have studied the incidence of tinnitus in the different forms of ear disease causing deafness. We know how to estimate the timbre of the tinnitus, and measure its loudness. We know how to measure the hearing capacity and have discovered several useful tests for aiding differential diagnosis in deafness and in tinnitus; but there are many instances in which deafness or tinnitus or both occur without any history of a prior inflammatory episode, without a family history, without any acoustic trauma, any fatigue or poisoning, or any abuse of drugs; and at autopsy, if we are fortunate enough to obtain one, we view only the end-results, we learn nothing as to causation.

*Read at the joint meeting of the Section on Otology, New York Academy of Medicine, and the New York League for the Hard of Hearing at the latter's Fortieth Anniversary celebration, Roosevelt Hotel, New York, N. Y., April 19, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 17, 1950.

Most people with deafness and tinnitus appear otherwise perfectly healthful. They seldom reveal vitamin deficiencies, or obvious disturbances in their mucous membranes, eyes, locomotor or cardiovascular systems. They often suffer from no apparent disease whatsoever. Maybe the only detectable abnormality is that one ear is deafened, or entirely deaf, or that there are head noises in one or in both ears. In some instances both ears are severely deafened (even totally). People who suffer from deafness associated with severe tinnitus are usually very sensitive, tense or "nervous." They are often called "the ulcer type." It is this mysterious tinnitus and deafness often seen, the origin of which has up to now eluded medical science, which we will discuss in this paper, and for which we believe we have discovered a cause, a cause which should be preventable, and even controllable. Please note that we say "a cause," not "the only cause."

If deafness is sudden and total it is customary to "guess" that it is due to a vascular accident. By vascular accident is meant a hemorrhage or bleeding into the tissues from a ruptured blood vessel. Without autopsy vascular accidents are difficult to prove; in fact, rarely have been proven in these cases. Moreover, in the types of deafness we are here discussing the deafness may not be sudden, and generally at least for a long time not total. Such patients as a rule show no sign or symptoms of disease or loss of function of any kind in any of their sense organs except the ear, in spite of the fact that many of them have been examined at the time or immediately after the tinnitus or deafness was first noticed. At one time or another they may have had vertigo, nausea and vomiting, but this may always be traced to the vestibular end-organ. The majority of these patients are not old persons, although these symptoms are not uncommon in old people. Arteriosclerosis, though it is sometimes present, is probably not prominent in the etiology. Arteriosclerosis frequently occurs without any tinnitus and deafness. These types of tinnitus and deafness also occur in people with various other bodily disorders and diseases. The underlying etiology for the disorders in other organs is often the same as that in the ear.

For instance, in our experience sudden deafness and tinnitus occur quite frequently with severe liver disturbances, and with duodenal ulcers and ulcerative colitis.

The number of discernible factors underlying and conceivably common to many diseases as well as to various disturbed psychosomatic states in otherwise healthful people is limited. What are some of the most commonly present factors which deserve consideration? Eliminating undiagnosed inflammatory destructions, poisons, blood dyscrasias, and trauma, there remain outstanding only the neurovascular, psychic, and nutritional disorders. The last may be disregarded as a common factor here because they are rarely encountered in these people, at least to no greater extent than in those with no tinnitus and no deafness. It is impossible to separate the psychic and neurovascular factors because they are often closely integrated, and, therefore, they should be, and herein will be, considered together. The question then is, how can psychoneurovascular factors cause the type of tinnitus and deafness we are here considering?

The general conception of the effects of emotional disturbances is that they may complicate the symptomatology of any disorder by their effect upon the psychological behavior of the patient, but that any effects that they may have upon the tissues of the body are transient and of no importance from the standpoint of a primary cause of disease, *i.e.*, that psychical disturbances in the average patient are "overlays" rather than the cause of disease or symptom. We believe this concept is not in accordance with the facts.

What do we mean by "emotion"? The dictionary definition is misleading. It takes no cognizance of unrecognized (subconscious) emotional states. Strictly speaking, emotion is a psychological and bodily state that in greater or less degree accompanies all mental and physical activity. Even slight emotion is accompanied by a physiological change. When it is extreme we can recognize it by flushing or blanching of the face, dilatation of the nares, trembling, weeping, a display of temper, hysterics, etc. We believe with Wolf, Cannon, Lewis

and others that there are often present less obvious changes in the circulatory, respiratory or muscular systems.

Psychologically speaking, there is an "effect" or "feeling tone" that accompanies all sensation. When the "feeling tone" is exaggerated by ideation (thoughts or ideas, conscious or unrecognized) it is called "emotion."*

Failure to act or overacting emotionally to the environment are factors which have been shown to lie at the root of many so-called "functional disorders." Also they may be largely responsible for the time of onset, severity, duration, recurrance, variation, and ultimate outcome of various diseases, and even for the instigation of many pathological states.

Profound changes occur in both man and beast from fear, rage, excessive fatigue, insecurity, longings, unhappiness, bereavement, despair or frustration through activity of the autonomic (involuntary) nervous system; both the parasympathetic and the sympathetic divisions are involved in these changes.

Under emotional stress blood pressure variations, flushing of various areas of the skin, blanching, fainting, nausea and vomiting and involuntary dilatation of the sphincters are observed, and are largely parasympathetic reactions. The involuntary or autonomic nervous system, whether activated directly or through the mediation of the blood stream, can affect the functions of almost every tissue or organ in the body whether it be in health or in disease, and whether or not the blood pressure is high or low, or average normal.

Both efferently and afferently there are intimate connections between the autonomic and central nervous systems. The hypothalamus is the chief integrating center for the autonomic impulses, and it has connections with the thalamus and the cerebral cortex. The cerebral cortex influences the autonomic nervous system in both of its divisions, and both divisions are widely distributed in the body; to the skin,

*This definition of emotion was evolved after consultation with Dr. L. Vosburg Lyons, of New York, N. Y.

blood vessels, viscera, reproductive mechanisms, secretory and excretory organs, endocrine glands, and the sense organs.

The ultimate reaction of so-called emotional episodes upon the tissue cells is caused by the chemical mediators adrenalin and sympathin called forth by sympathetic stimulation, and by acetylcholine released by parasympathetic action, and also by the hormones of various glands of internal secretion either directly or indirectly.

Whether the resulting clinical symptoms or lesions are more sympathetic or more parasympathetic depends upon the relative stimulation of these two divisions of the autonomic mechanisms, and upon the hyper- or hypoactivity of the tissue cells, or the nerve supply to the organ affected.

The psychosomatic effects of emotions upon many disorders and diseases are legion, but when an incidental illness coincides in time with severe emotional shock the illness is made worse and a relapse occurs, and the two together may be more than the patient can stand.

Frequently the emotional factors are overlooked because the physician assumes that the patient will bring them to his attention if they are important. As a matter of fact we all of us often have more powerful "forgetories" than "memories," and so we do not bring them to the attention of our physician.

Old memories aroused by some appropriate stimulus can bring back the intense emotions originally associated with them. Such memories may be subconscious and still bring back the emotional reactions and their concomitant repercussions upon the autonomic neural mechanisms. This may occur during sleep. The patient may then attribute his disturbed feelings to some recent happening, and not realize its true source, an episode in the past. When there is an autonomic hypersensitivity, or an already damaged organ, the renewal of emotional tension or imbalance can lead to exacerbations of the lesion and to an increase in symptoms.

How may we decide whether or not emotion is a significant factor in "organic," or in so-called "functional disturbances"

of the ear? Of course one preliminary is to exclude prior organic disease of the auditory apparatus. This is often difficult, and moreover both organic and functional disorders may arise from similar emotional states, and both may exist at the same time.

Important criteria for correlating emotional and somatic factors are:

1. Establish a time relationship between the emotional episodes and the onset or exacerbation of symptoms. (We are herein concerned with tinnitus and deafness in otherwise apparently healthy persons.)

2. If no correlation is remembered or recognized, or no memory remains of the emotional upsets, yet the patient is and has been hypersensitive to emotional stimuli, then it is highly probable that emotion was a factor in the etiology. There is *nothing else more probable.*

How do episodes of hypersensitivity, or as they are commonly called, "states of emotional instability," interfere with normal metabolism and bring about irritation, diminished function and even degeneration of the auditory (and vestibular) neural mechanisms? An outline of what often probably happens may be set forth as follows: Whether as a result of a present or prior illness hypersensitiveness of an organ results in an over response to sympathetic stimulation. The blood vessels in the organ over contract, or over relax, during too long periods of time. Over contraction cuts down the calibre of the small arterioles and venules, slows the blood stream and furthermore it has been shown that with sympathetic stimulations blood sludge is created. This further interferes with the passage of blood through the swollen vessels. If severe these reactions may shut it off completely from a given area.

The stasis deprives the tissues of oxygen, sugar and other metabolites, and interferes with the removal of waste and heat from the tissues. Oxygen is one of the most important blood borne materials, and even a slight local oxygen deficiency will

upset the healthy functioning of the tissues and eventually cause degeneration in the more susceptible elements therein. Unless collateral circulation promptly comes to the rescue the following events have been observed to take place:

Decreased blood volume alone, even following a loss of 300 to 500 cc. in blood donors, is sufficient to cause tight contractions in first one and then other sets of arteries, arterioles, capillaries, postcapillaries and venules observed in the ocular conjunctiva. Only a few larger vessels may remain open. Similar reactions occur in the blood vessels of other tissues. There may be the initial signs of shock, such as profuse sweating, chilliness and giddiness. If no counter stimuli or inhibitions intervene prolonged contractions have been observed, and the affected tissues are literally asphyxiated for considerable periods of time (see Fig. 1).

After traumatic shock (even without loss of blood) and in all severe infections and toxic diseases, thick agglutinated masses have been observed to form by the sticking together of the erythrocytes (red blood cells). The masses are called "sludges." Inserting a fairly large needle into the body is sufficient to produce sludging of the blood, even causing the patient to react to a slight surface irritation (*i.e.*, electric shock). Even a provocative remark may cause a contraction of the smaller vessels often followed by sludging of the blood.

Sludges eventually find their way into the general circulation, and before they are destroyed, as eventually happens in the liver and spleen, they may have caused profound changes in some areas of the body. They are most easily observed in the smaller arteries and venules in the ocular conjunctiva of the unanesthetized patient and here provide us with a fair sample of all of the circulating blood.

Using binocular microscopes with high magnification (50 to 100×) and brilliant illumination obliquely directed, with adequate thermal protection, we can focus our microscope on the vessels in the bulbae conjunctiva to study the normal and pathological blood flows. We have been intensely studying the degrees of sludging, its rigidity and retarding effects upon

Fig. 1. Sketch of a normal conjunctival field × 100 approx.

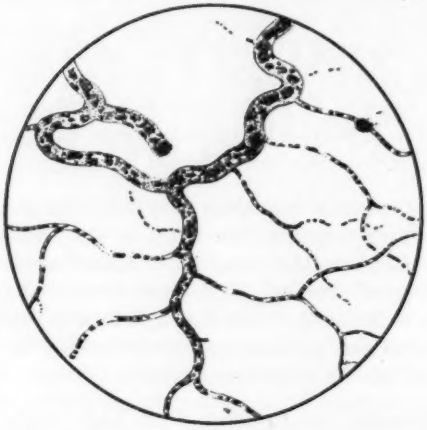

Fig. 2. Sketch of the same conjunctival field after cervical sympathetic trunk stimulation or injection of intravenous epinephrin. The smudge, upper right, is a small hemorrhage.

Reprinted from the Proceedings of the Society of Experimental Biology and Medicine. Blood Sludge from Sympathetic Stimulation, by E. P. Fowler, Jr., 72:592. 1949.

the circulation, the state of the vessel walls, and their pathologic reactions to stimuli.

Knisely* has demonstrated that in perfectly healthy persons (and in the so-called "lower animals") the blood flow is normally laminar and streamlined. No stacking (Rouleaux) or clumping of the blood cells was obeserved, except in abnormal conditions.

In health, and if no local trauma has occurred, the red cells are not coated with a sticky precipitate and show no tendency to adhere to each other. Neither white nor red cells adhere to the smooth and clean inner walls of the small vessels. Around the axial stream of cells is a peripheral concentric layer of blood plasma. Each succeeding layer toward the periphery flows more slowly than the one immediately inside it, and each layer is exactly one red cell thick. The diameters of the narrowest and more powerfully contractile vessels in the tissues (the arterioles, terminal arterioles, contractile sphincters, etc.) may remain constant or change from moment to moment as is necessary for physiological adjustments in response to changes in the equilibrium between "constrictor and dilator" substances reaching the vessel wall from *1.* blood within the vessel, *2.* cells surrounding the vessel, and *3.* nerve endings, in, upon, or near the vessel wall. Temperature changes in the surrounding tissues also affect the contractile elements.

Further, according to Knisely and his co-workers, every homeostatic dilatation of any group of small vessels produces hydrodynamically an increase in the rate of blood flow through these vessels and an increase in the rate of supply of oxygen and metabolites, and in the removal of wastes.

The blood normally flows so rapidly in most arterioles and venules (diameters 60 to 120 μ) that the individual red cells cannot be seen. This invisibility serves as a fairly useful criterion for judging the adequacy of the speed of flow through most of the open-tissue capillaries. If unagglutinated blood cells cannot be seen under high magnification there is no

*Knisely, M. H.; Eliot, T. S.; Block, E., and Warner, L.: Science, 106:2758, 1947.

visible hemoconcentration taking place in the capillaries. In other words, the capillary walls are not leaking blood fluid.

Every blood cell, red and white, passing through open arteriole terminals is forcibly distorted because it cannot be shoved through without being elongated or folded. In the capillaries there is more room, because the diameters vary from a little less than one to as much as at least two and one-half times the diameters of the red cells, and true capillaries can usually dilate without losing their tonus.

Knisely believes that almost every arteriole to capillary or sinusoid to venule pathway can contract tightly shut throughout its length, its lumen then being zero, and that when no blood can enter, in some or all of its parts dilatation may take place to the maximum permitted by the surrounding supporting connective tissues, normally not over two and one-half times the diameter of the red cells. Forcibly overdilated capillaries permit both blood plasma and protein to leak into the sourrounding tissues. *No severely ill patient has been observed who did not have sludging of the blood and visibly damaged vessel walls.* "Under all conditions, in health and in disease, the arterioles and capillaries are a perpetual bottleneck in the vascular system. These bottlenecks are increased by the resistance of sludged blood to its own passage, and the rate of flow is forcily reduced in all the capillary beds of the body."

In some organs there is such a wide margin of safety that moderate contraction of the vessels or moderate sludging causes no detectible symptoms, but in the auditory neural apparatus of the cochlea the vessels are terminal, there is no collateral circulation to come to the rescue when a branch of the cochlear artery is obstructed. It is reasonable, therefore, to suggest that contractions or sludging or both could be, even often are, disastrous to the neural elements involved. It should not be surprising if irritation so caused resulted in tinnitus, and if prolonged eventually resulted in an irreversible degeneration in the neural tissues; the auditory ganglion cells, the nerve fibres, and their insulating sheaths. Such degenera-

tions are regularly found postmortem in patients with these types of tinnitus and deafness. It should not be surprising that emotional episodes, remembered or forgotten, suppressed or evaded, are usually a determining factor in emotional problems, and that they can set up the reactions described. We have frequently been permitted to correlate them with the onset of, and with variations in tinnitus and deafness.

We wish to point out that vessel contraction, sludging, and the accompanying phenomena can affect not only the neural elements in the ear (and elsewhere in the body) but also non-neural tissues, even the bones, and here we have a promising lead for investigations in connection with the etiology of otosclerosis.

Sludge is encountered in many diseases and disorders of the body. How does it happen that it is not always accompanied by tinnitus and deafness? One reason is that there are many kinds of sludge, and sludging phenomena, and varying time factors involved; moreover, the prior presence of sludging, its degree and the prior stability and health of the organ are of importance. In studying the vessels and the sludging phenomena, the size, shape, consistency, compressibility, spacing, and rate, and continuity of flow are important. Heredity and previous trauma from injury or disease will determine whether sludge lingers and produces local disorders, or pours through the vessels freely to organs where microscopic blocks may produce no symptomatology.

Now that we have learned something about the phenomena accompanying sludging we know in which direction our investigations and treatments should be directed. We already possess one means for controlling the phenomena, and more will be forthcoming. They will be discussed in a future report.

It is high time that the otologist should not be satisfied with a diagnosis of the pathology, or of the infecting agent, or of the mechanical cause of an ear disease, or even with a study of dead tissues under the microscope. He should investigate the biological causes which made the patients susceptible to

them. He should study pathology in the living patient. The closer he gets to a study of living individual cell physiology and pathology the nearer he will get to the truth.

We feel that the observation of changes in the blood itself in cases of deafness and tinnitus, and the alleviation of tinnitus by intravenous procaine (which had previously been shown to break up blood sludge) are steps in the right direction. We have, of course, not proven incontrovertibly that emotionally induced blood sludge produces tinnitus and deafness, but there certainly seems to be strong evidence that this may be the case in some individuals.

THE CYTOLOGIC DIAGNOSIS OF PRIMARY BRONCHOGENIC CARCINOMA.*†

JOHN J. O'KEEFE, M.D.,
Philadelphia, Pa.

In the universal endeavor to eradicate cancer, the literature[6,7,11] is abundant in its recordings of efforts directed towards the development of better diagnostic methods. The motivating force being that only by the establishment of such procedures will early diagnoses be feasible and, consequently, cures possible.

The summation of facts gathered from the recent literature[1,3,12] indicates that primary bronchogenic carcinoma is predominantly a disease of white men; its greatest incidence is between the ages of 45 and 60; it has a definite predilection for the right lung; the average length of life in cases in which the patient is not amenable to surgical cure is 12 to 18 months. Its cardinal symptoms, in the order of frequency of occurrence, are recorded as cough, pain in the chest, expectoration and dyspnea. The histopathologic picture presented is predominantly that of epidermoid carcinoma, with an occasional report of adenocarcinoma.

Clinical manifestations of this disease usually are not evident until after the primary lesion has undergone moderate growth. The majority, of squamous cell type, manifest symptoms through invasion of regional tissue, producing secondary factors, such as erosion and ulceration of the mucosa, bronchial obstruction with retained secretions, secondary infection, atelectasis and/or compression of one of the larger pulmonary vessels. Tumors originating in the extreme periphery,[8,9] the "alveolar cell tumors," are more apt to produce

*Presented as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1950.

†From Department of Bronchoesophagology, Jefferson Hospital, Philadelphia, Pa.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 1, 1950.

symptoms referable to the pleura, or to remote organs by metastases. Too often, both types produce metastatic lesions before adequate consideration has been given the symptoms caused by the primary lesion of the lung.

TABLE 1.

SYMPTOMS ON PATIENT'S ADMISSION TO THE HOSPITAL.

	Per Cent
Cough	76.3
Thoracic Pain	62.5
Sputum	56.4
Dyspnea	39.6
Hemoptysis	38.9
Loss of Weight	36.6
Loss of Strength	32.0
"Cold"	26.7
Night Sweats	7.6
Hoarseness	7.6
Wheezing	2.2

The development and presence of detectable physical signs are evidences of advanced growth and almost invariably preclude the possibility of operability.

The literature varies somewhat in its recorded statistics,[2,3,5,6,12] but such variability only lends emphasis to the facts *1.* that the symptoms of early bronchogenic carcinoma follow no stereotyped pattern, *2.* that every symptom referable to the chest reported by the patient should be considered as organic in origin, and *3.* that investigation should be pursued until an accurate diagnosis is attained.

DIAGNOSTIC METHODS.

Roentgenology: The Roentgenogram is probably the most valuable single means of obtaining an early presumptive diagnosis of bronchogenic carcinoma. It is well to bear in mind that early developing densities in the lung fields follow no form or pattern; their outlines are indistinct and their margins irregular; small shadows, situated peripherally, with a tendency to clear, only to recur, are in all probability neoplastic; and that the commonest Roentgenologic manifestation of total bronchial obstruction[4,10] is atelectasis of the segment, lobe or lung tissue distal to the tumor. The adjunctive use of

such special procedures as planography and bronchography may add corroborative evidence to the presence of suspected intrabronchial or extrabronchial tumors.

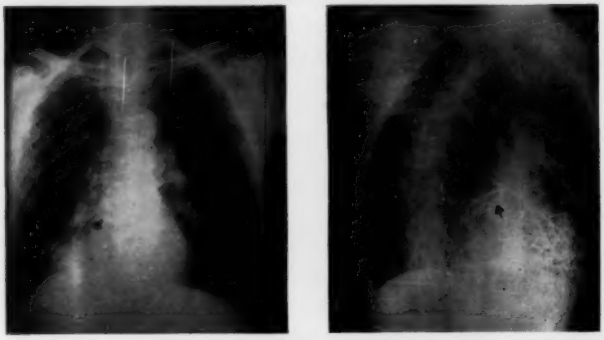

Fig. 1.

A.
Showing an area of segmental atelectasis of the lower lobe of the right lung.

B.
After instillation of iodized oil, showing the bronchial obstruction which caused atelectasis. Cytologic study of bronchoscopically removed secretions revealed carcinoma cells.

Bronchoscopy: The indication for diagnostic bronchoscopy consists in the presence of bronchopulmonary symptoms, the explanation of which is incomplete or unsatisfactory. This study should be made in all cases of suspected bronchogenic carcinoma, as evidenced clinically by persistent cough, pain in the chest, wheezing or hemoptysis, in cases in which the presence of neoplasm is based upon the Roentgenologic findings, and in those in which obscure pulmonary symptoms are manifested.

Views of bronchogenic carcinoma obtained bronchoscopically are variable. The tumor is usually nodular, is soft and red, often ulcerated, and bleeds at the slightest trauma. Such lesions offer no difficulty to the securing of a positive biopsy diagnosis. Other lesions, especially those situated at the extreme periphery or in the upper lobe, are infrequently capable of visualization bronchoscopically. It is because of this type of lesion that further means of diagnosis must be devised and

employed in order to secure an earlier positive histologic diagnosis.

Aspiration Biopsy: Although approved of initially, the associated danger of implant metastasis in the tract of the aspirating needle has fostered much controversy as to the practicability of its continued use.

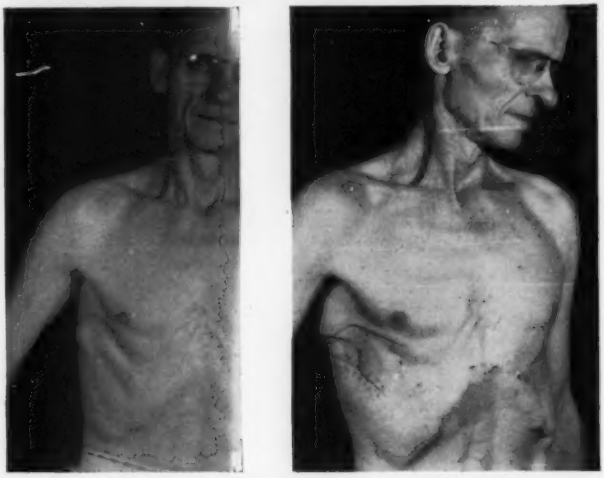

Fig. 2.

A. Photograph of pneumonectomized patient, showing development of implant metastases in the chest wall at the exact site of aspiration for biopsy.

B. Photograph of the patient after excision of the lesion.

Exploratory Thoracotomy: To balance the scale, the practice of exploratory thoracotomy has gained universal widespread favor; however, even this practice has its shortcomings, for at the time of thoracotomy the nature of a small, centrally placed lesion is indistinguishable from an area of pneumonitis, an abscess or an anthracotic or silicotic nodule.

Cytologic Method: Most recently developed, and of excellent promise, is the practice of cytologic study of bronchoscopically removed secretions. This procedure has added a

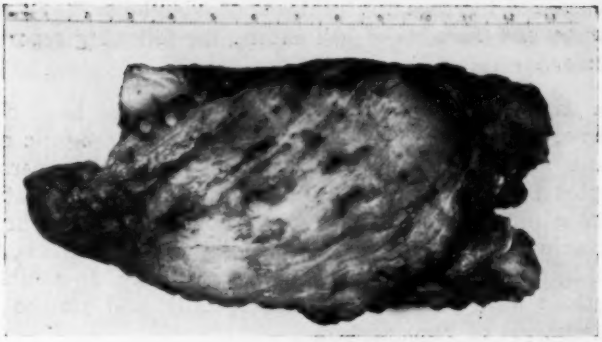

Fig. 2. C.
Photograph of the tumor removed from the chest wall.

high percentage of positive results in the early diagnosis of bronchogenic cancer. It is of particular value in the study of those lesions that are peripherally situated and bronchoscopically inaccessible. Its practice, too, is free of the dangers, real or imaginary, that are integral parts of the technique of aspiration biopsy and thoracotomy.

CLINICAL STUDY.

The stimulating influence in the development of this method of diagnosing carcinoma originates in the staining technique as devised by Papanicolaou.[11] Its application to carcinoma of the lung was initially reported by Herbut and Clerf,[7] in 1946. These observers, reporting on 30 consecutive cases of lung cancer, state that "cancer cells were demonstrated in 22 cases, or 73 per cent. In the same series a positive morphologic diagnosis, from a study of tissue removed endoscopically, was obtained in 11 cases, or 36 per cent. Cancer cells were present in secretions from seven cases in which bronchoscopy was negative."

Consideration and study of this report strongly suggests that in the endeavor to diagnose cancer early, a method of singular merit has been devised. Further, it leads one to query, "Will the diagnosis so obtained be early enough to

enhance and increase the incidence of operability?" To this question, and thoughts of like nature, the following report is submitted:

In the ensuing three years, 1946-1949, there have been collected 307 consecutive cases of primary bronchogenic carcinoma. Each of these has been studied in the generally accepted present-day methods, consisting of a consideration of the symptoms and physical signs, Roentgenography, bronchography, bronchoscopy, the histologic study of tissue secured by biopsy, and by exploratory thoracotomy. In addition, secretions were obtained bronchoscopically, and stained by the technique of Papanicolaou.

Of the 307 proved cases, in 272, or 88.6 per cent, a diagnosis of carcinoma was made by cytologic examination of bronchoscopically aspirated secretions. In this same group, bronchoscopy was positive by biopsy in 104, or 33.8 per cent; bronchoscopic evidence of altered morphology (stenosis, deformity, fixity) was noted in an additional 87, or 25 per cent. Of paramount significance is the fact that in 94, or 30.6 per cent of cases, in which the bronchoscopic findings were entirely normal, cytologic study of bronchial secretions was positive for neoplastic cells.

TABLE 2.

307 PROVED CASES OF CARCINOMA OF THE LUNG.

	Number	Per Cent
Diagnosis by Cytologic Study	272	88.6
Diagnosis by Bronchoscopic Biopsy	104	33.8
Diagnosis by Altered Morphology	87	25.0
Cytology Positive, Bronchoscopy Negative	94	30.6

The impressive facts in the analysis of these figures is *1.* that by the cytologic method alone, 272, or 88.6 per cent of cases were diagnosed positively, and *2.* that in 94, or 30.6 per cent of cases, although bronchoscopy was entirely normal, the bronchoscopically removed secretions were positive for neoplastic cells. Herein, then, lies a diagnostic method that both elicits a high percentage of positive results and adds an appreciable margin of positive results in otherwise negative cases.

It is generally accepted that the presence of carcinomatous mediastinal and/or bronchial nodes encountered in the process of pulmonary resection for bronchogenic cancer, mitigate against the probability of cure. Using this as a means of evaluation of the cytologic study of bronchial secretions as a method of diagnosis of lung cancer, 69 such consecutive total pulmonary resections were analyzed. Of the 69, bronchoscopy was negative in 25, and positive in 44. Of the 25 negative cases, the cytologic study was negative in seven and positive in 18. In the same seven there were no lymph nodes noted, and in the 18 only two cases had nodes. The entire 25 bronchoscopically negative cases may be said to have a good prognosis. Of the bronchoscopically positive group, cytology was positive in 39, and negative in five. Lymph nodes were present in 19 of the 39 positive cases, and in four of the five negative cases. The outlook for cure has fallen appreciably.

TABLE 3.

TOTAL PULMONARY RESECTIONS — 69.

Bronchoscopy	Cytology	No.	Total	Lymph Nodes	Total
Negative	Negative	7		0	
Negative	Positive	18	25	2	2
Pos. Biopsy	Positive	21		11	
Pos. Deformity	Negative	5	44	4	23
Pos. Deformity	Positive	18		8	

The point of pertinent interest in the study of Table 3 is that the bronchoscopically negative cases indicate a far better prognosis than do the bronchoscopically positive ones and that only by obtaining patients before deformities occur is surgical resection curative.

SUMMARY.

Primary bronchogenic carcinoma is reviewed. A summation of its characteristics is presented, and the generally accepted methods of diagnosis are examined. The most recently developed diagnostic procedure, cytologic study of bronchoscopically removed secretions, is clinically evaluated.

An effort is made to show that this method is efficient in obtaining a positive diagnosis of lung cancer in a large percentage of cases, and that it adds an appreciable margin of positive results in otherwise negative cases.

Further, it is shown that diagnoses obtained before bronchial deformities occur increase the possibility of operability and indicate a more favorable prognosis.

BIBLIOGRAPHY.

1. Holinger, P. H.; Hara, H. J., and Hirsch, E. F.: Bronchogenic Carcinoma: An Analysis of 175 Proved Cases. *Ann. Otol., Rhinol. and Laryngol.*, 54:5, 1945.

2. Ochsner, Alton, and DeBakey, Michael: Surgical Considerations of Primary Carcinoma of the Lung. *Surg.*, 8:992, Dec., 1940.

3. Perrone, J. A., and Levinson, J. P.: Primary Carcinoma of the Lung (Report of 115 Cases, 38 Autopsies and 77 Bronchoscopic Biopsies). *Ann. Int. Med.*, 17:12, July, 1942.

4. Clerf, L. H.: Carcinoma of the Bronchus. *Radiol.*, 28:438, 1937.

4. Ochsner, Alton, and DeBakey, Michael: Significance of Metastasis in Primary Carcinoma of the Lungs. *Jour. Thoracic Surg.*, 11:357, Apr., 1942.

6. Overholt, R. H.: Curability of Primary Carcinoma of the Lung: Early Recognition and Management. *Surg., Gynec. and Obst.*, 70:735, 1940.

7. Herbut, P. A., and Clerf, L. H.: Bronchogenic Carcinoma; Diagnosis by Cytologic Study of Bronchoscopically Removed Secretions. *Jour. A. M. A.*, 130:1006, Apr., 1946.

8. Herbut, P. A.: Bronchiolar Origin of "Alveolar Cell Tumor" of the Lung. *Am. Jour. Pathol.*, 20:911, 1944.

9. Herbut, P. A.: "Alveolar Cell Tumor" of the Lung; Further Evidence of Its Bronchiolar Origin. *Arch. Pathol.*, 41:175, Feb., 1946.

10. Farrell, J. T., Jr.: Diagnosis of Bronchial Carcinoma. *Radiol.*, 26:26, 1936.

11. Papanicolaou, George N., and Cromwell, Henry A.: Diagnosis of Cancer of the Lung by the Cytologic Method. *Dis. Chest*, 15:4, Apr., 1949.

12. O'Keefe, John J.: Primary Bronchogenic Carcinoma. *Arch. Int. Med.*, 82:345. Oct., 1948.

255 South 17th Street.

AUREOMYCIN IN TREATMENT OF OTITIC AND OPHTHALMIC HERPES ZOSTER.

EDWARD W. GANS, M.D.,
Oakland, Calif.

The treatments of herpes zoster have been numerous, each having an apparent value but none specific enough to be completely relied upon. Becker and Obermeyer[1] state that so many treatments of herpes zoster have been recommended that it is questionable whether many of them have any specific effect. O'Neill,[2] in listing some of the various treatments, remarks that obviously many of the therapeutic suggestions for treating a disease of unknown but suspected virus origin border on the irrational. We see case after case resolve with only symptomatic and supportive therapy, which makes one wonder, during treatment, whether there is anything even bordering on a specific approach.

The probability of a virus being the etiological agent in herpes zoster has long been held. Sutton and Sutton[3] state that a virus as the causative agent is strongly suspected and quote literature showing the possible virus etiology as well as its relationship to varicella. Treatments listed by them were legion, varying from local therapy designed to prevent secondary infection and medication to relieve pain, to specific therapy such as deep X-ray to the affected ganglion, intravenous iodides and pituitary extract, a full surgical dose of the extract of the posterior portion of the gland. Injections of alcohol into the intercostal nerves after a preliminary injection of novocaine, although claimed to be an excellent treatment, have not proven effective in every case. Cevitamic acid by mouth and intravenously, diphtheria antitoxin in doses of 5,000 units given once and perhaps a second time after two days have also been advocated.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 2, 1950.

In his discussion of ophthalmic herpes zoster, Duke-Elder[4] relates that the herpetic neuralgia can be the most intense of all pains, attacking with cruel insistence day and night for weeks and aggravated by every contact or draft of air. He added that definite ocular complications arise in 50 per cent of cases at any time during the eruptive stage, or some weeks after the rash has subsided. These complications are keratitis, scleritis, iridocyclitis, ocular palsies and optic neuritis.

Chronic otalgia may result from otitic herpes requiring sectioning of the sensory roots of the geniculate ganglion to obtain relief. Lederer[5] remarks that since the Vth, VIIth and VIIIth nerves may suffer at the same time, vestibular symptoms are marked in some cases with secondary involvement of the auditory part, or vice versa. The facial nerve and its branches may also be affected, resulting in paralysis from which the patient often has difficulty in recovering. Sometimes the deafness remains permanent.

We can see that herpes zoster about the head and face can be very severe in its symptoms and complications. Medicine is, therefore, greatly in need of a reliable, specific treatment that will shorten the course of this disease, decrease the complications and bring about an early relief of pain.

With the advent of aureomycin and its apparent specific action against certain virus infections, the question of its efficacy in the treatment of herpes zoster arises. When confronted with a case of otitic herpes and a VIIth nerve paralysis, the Ramsey-Hunt syndrome, it occurred to me that treatment with aureomycin was well worth trying. The possibility of serious sequela far overshadowed the cost of treatment, if it were at all successful. The apparent success with this case led us to use aureomycin in the treatment of all cases of herpes zoster seen in the eye, ear, nose and throat department during the next several months with gratifying results.

Lately Maxwell Finland, *et al.*,[6] presented 25 cases of herpes zoster treated with aureomycin, and concluded that the drug appeared to halt the progress of the disease and bring about a rapid healing of the lesions. They noted that pain was

relieved after the first day of treatment and that cases treated early responded much more rapidly and completely; these were also our observations. We also noted, as did they, that ophthalmic cases did not so readily tend towards corneal scarring. Due to the high cost of the drug, our daily dosage was half of what the above named men used, and the length of treatment shorter; yet we apparently obtained good results. It is interesting to note that following treatment all the patients felt that the results were well worth the cost. One man, Case 6, would consent to only one day's supply but returned the next day for more because of the relief he had received.

The dosage of aureomycin administered was 500 mg. every six hours for adults, and the treatment continued for two to three days. The one child seen received 250 mg. every six hours for three days. In conjunction, vitamin B, physiotherapy and iodides were used as well as protective medication and analgesics. Coley's vaccine was used in one case.

Below are presented six cases treated between April, 1949, and January of 1950. All of these cases were seen relatively early.

Case 1: B. H., white female, age 17 years, developed a painful right ear, followed the next day by a stiffness and paralysis of the right face. She was first seen three days after the onset, and at that time an herpetic-like lesion was noted in the concha of the right auricle, and the entire auricle was tender and sensitive; otherwise the ear was objectively negative. The face showed a complete lower neuron N VII paralysis. Treatment consisted of vitamin B complex by mouth, aureomycin, 500 mg., orally every six hours, and physiotherapy. When seen three days later she stated that her ear began to feel much better on the second day and she was able to sleep on it that night. The herpetic lesion was completely dry and healed. The patient finished her physiotherapy treatments 13 days after the initial visit, at which time her facial paralysis was completely cleared, and when contacted two months later there had been no further trouble or recurrence.

Case 2: P. L., white male, age 65. First seen by me, complaining of a left earache and pain over his left neck. Impacted cerumen was removed from the ear canal and examination revea'ed no positive findings. Aspirin and local heat were advised. Three days later he was seen in dermatology with a typical herpes zoster over the left sca'p and forehead, complaining of severe pain. Four days of treatment with sedatives, sodium iodide, 1 gm. I. V., and thiamin chloride, 100 mg. daily, caused no change, and due to involvement of the eye he was referred to the department of ophthalmology for care. No corneal lesions were found, and the patient was placed on aureomycin, 500 mg. every six hours, and continued on thiamin chloride orally. When seen 48 hours later there was

a marked decrease in pain and less edema of the eyelid. Aureomycin was discontinued after the third day, and by the sixth day following institution of aureomycin therapy, the lesions showed a marked clearing over the entire involved area. Pain had completely gone except for some mild intermittent shooting pains over the left scalp area. Fifteen days after his initial visit K. P.'s were noted on the corneal endothelium and he was given three 100 mg. injections of thiamin chloride, intramuscularly. He was seen regularly thereafter; the skin lesions cleared rapidly and when last seen, seven months following his original complaint, the K. P.'s had disappeared and he was perfecty clear of any residual of infection.

Case 3: R. B., white male, age six years, developed an acute upper respiratory infection followed in three days by an acutely painful right ear and discharge. Treated in pediatrics with sulfadiazine, the upper respiratory infection was successfully controlled, but severe pain developed in his right ear, followed by a vesicular eruption about the auricle. First seen by me in ear, nose and throat four days following ear pain, and two days after vesicles were noted. Examination revealed a vesicular eruption about the vestibule of the right external canal, the concha, tragus and preauricular area. The external canal was eroded and tender, and the tympanic membrane was found to be intact and injected, but neither bulging nor retracted. A gauze wick was inserted and continuous moist compresses of aluminum subacetate solution prescribed. Aureomycin was prescribed, 250 mg. every six hours. When seen the following day, he was much improved. Although there were a few vesicles appearing in the concha, the pain was markedly decreased. He continued to improve, and aureomycin was discontinued after the third day. The compresses were continued until last seen, eight days following his first aureomycin medication; the lesions were completely clear, the canal healed and the tympanic membrane normal in appearance.

Case 4: G. D., white male, age 35. First seen in emergency ward by me at night, with vesicular herpetic lesions over his left forehead and upper eyelid, complaining of severe pain. He stated that six days previous dust had flown into his eye and he had been exposed to an electric flash. Two days later a man at his plant removed a foreign body from his eye, and the following day he developed blisters over his forehead and eyelid. There was not much pain at that time. He was started on aureomycin, 500 mg. every six hours, sulfathiazol ophthalmic ointment, and the eye dilated with homatropine. Codeine and salicylates were prescribed for pain, and he was referred to the department of ophthalmology for further care. The following day examination of the eye was negative for any corneal involvement. All pain had ceased on the second day of treatment. By the third day the vesicles began to resolve, and cells were noted in the anterior chamber; also corneal infiltrates made their appearance. Aureomycin was discontinued after the third day, and by the fourth day the vesicles had almost disappeared; the corneal infiltrates were more pronounced and he was given Coley's vaccine Mii. Six days later, K.P.'s were found behind the infiltrates, and occasional cells in the anterior chamber were seen. Coley's vaccine was again given. Two days later he was given his third Coley's injection; the K. P.'s had gone and the anterior chamber had cleared. When last seen 17 days later, there were still a few infiltrates in the cornea.

Case 5: M. S., white female, age 30 years. When first seen by me, stated that she had awakened that morning with a severe deep pain in her left ear. She stated that she had had shingles on her thigh six years before. Examination revealed an intact normal tympanic membrane. There were a few herpetic-like lesions about the vestibule of the canal and in the

concha. The patient refused aureomycin therapy due to the expense, so was given codeine and aspirin for pain and advised to use hot compresses. Three days later she returned, still having the severe pain, only partially controlled by the codeine. The vesicles were the same in number but larger in size. She had developed an acute catarrhal conjunctivitis of the left eye the night before. She was placed on aureomycin, 500 mg. every six hours, and sodium sulfacetamide ophthalmic drops, 30 w/v, and hot compresses to the eye. Two days later she called, stating that the pain was much improved but that she had developed nausea and vomiting with the last two doses of aureomycin. In all, she had retained five 500 mg. doses of the drug. When next and last seen by me five days following the beginning of aureomycin therapy, all lesions had healed, and she stated that the pain had eased within 24 hours after taking the aureomycin.

Case 6: E. B., white male, 43 years old. First seen in the department of ophthalmology with vesicular herpetic eruption over his scalp and forehead, and upper eyelid, complaining of severe pain over the involved area. First noted two to three days previously. No involvement of the cornea was noted. Aureomycin, 500 mg. every six hours, was prescribed along with crude coal tar topically and codeine and salicylates for pain. Aureomycin was continued for two days and when seen on the third he was subjectively improved. There was a marked edema of the upper lid but no pain, no corneal involvement and no increase in the lesions. He stated that the pain had gone after the first 24 hours. When last seen one week after his initial visit the lesions were almost cleared, and he had no subjective complaints. He did not report back for further follow-up or treatment so was considered cured.

SUMMARY AND CONCLUSIONS.

I have given a short resumé of the treatments of herpes zoster and considered the probable etiology of the disease. The gravity of this disease when affecting areas about the head and face is not to be denied. Any drug, therefore, that shortens the duration, relieves the pain and decreases complications is an important therapeutic addition. Six cases treated with aureomycin in the department of Ophthalmology and Otolaryngology of Permanente Foundation Hospital are presented. In all six cases there was rapid control of pain, early healing of lesions and a minimum of complications; only one case showing any residuum when last seen.

It is concluded, therefore, from these cases and others presented in the literature that aureomycin has a specific effect on herpes zoster infection.

BIBLIOGRAPHY.

1. Becker, S. W., and Obermeyer, M. E.: Modern Dermatology and Syphilology. Philadelphia, Pa.: J. B. Lippincott, 2nd Ed., 1947, p. 547.

2. O'NEILL, HUGH: Total Herpes Zoster of the Ophthalmic, Maxillary and Mandibular Divisions of the Trigeminal Nerve. *Arch. Ophth.*, 33:242, Mar., 1945.

3. SUTTON, RICHARD, and SUTTON, RICHARD, JR.: Diseases of the Skin. St. Louis, Mo.: C. V. Mosby Co., 10th ed., 1939, pp. 850-855.

4. DUKE-ELDER, SIR W. STEWART: Textbook of Ophthalmology, Vol. II. St. Louis, Mo.: C. V. Mosby Co., 1944, pp. 1902-1904.

5. LEDERER, FRANCIS L.: Diseases of Ear, Nose and Throat. Philadelphia, Pa.: F. A. Davis, 4th ed., 1943, p. 88.

6. FINLAND, MAXWELL, *et al.*: Aureomycin in Herpes Zoster. *N. Eng. Jour. Med.*, 241:1037-1046, Dec. 29, 1949.

Permanente Foundation Hospital.

ATYPICAL CASE OF FOREIGN BODY IN THE ESOPHAGUS.

ABDUL RASHID, M.D.,
Lahore, Pakistan.

An infant, aged five months, was admitted in the ear, nose and throat ward of the Mayo Hospital, Lahore, on Jan. 13, 1950. A history of his having swallowed a foreign body (metallic button) was available and this button was believed to have been put into his mouth by his brother.

Examination: The child showed signs of marked dyspnea and was in great distress, but he was able to take milk. There was recession of the intercostal, supra- and infraclavicular spaces; indrawing of the sternum, respiration was noisy, temperature was normal and pulse was 120 per minute.

Immediate tracheotomy was the only rational treatment at that moment, because of the great respiratory distress, and laryngoscopy had only a subsidiary value and was, therefore, postponed.

Low tracheotomy was done under local anesthesia with 0.5 per cent novocaine. One cc. of coramine was given as an analeptic. After tracheotomy, the breathing became quiet and easy, and the child looked comfortable. Further examination and treatment were postponed to enable the child to regain his strength.

After two days, screening showed opaque foreign body one-half inch above the jugular notch. Under general anesthesia through the tracheotomy tube, the larynx and upper part of the trachea were examined with the bronchoscope. A red swelling was seen distal to the vocal cords, obstructing the trachea, but no foreign body could be seen. Since as esophagoscope of a small size was not available, a bronchoscope was employed to explore the esophagus. The metallic button was visible just below the level of the cricoid with its base pushing the anterior wall of the esophagus forward and the vertical part directed backwards toward the posterior wall of the esophagus. The button was removed without much difficulty.

CONCLUSION.

This case is of interest in that although the foreign body was in the esophagus, yet there were no symptoms such as dysphagia. The only symptom present was respiratory distress. The button was placed in antroposterior position, the

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 28, 1950.

broad surface of the button had pushed the anterior wall of the esophagus and the thin posterior wall of the trachea forward, causing the respiratory distress. The absence of the difficulty in swallowing is accounted for by the position of the button. The milk could pass on either side of the vertical part of the button with no resultant dysphagia. Tracheotomy tube was removed after three weeks, and the child was discharged one week later.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YC and YC-7) (Veri-Small).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM; Model M-35; Model P-15.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Philip E. Meltzer, 20 Charlesgate, West Boston 15, Mass.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Green Briar Hotel, White Sulphur Springs, W. Va., May 9-10, 1951.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Secretary: Dr. C. Stewart Nash, 108 Medical Arts Bldg., Rochester, N. Y.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich.
Vice-Chairman: Dr. J. M. Robison, 1304 Walker Ave., Houston 2, Tex.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October 3-6, 1950.
Hotel Biltmore, New York, N. Y., January 8-11, 1951.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles, Calif.
President-Elect: Dr. Derrick Vail, Chicago, Ill.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 8-14, 1950.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. George E. Shambaugh, Jr., 55 E. Washington St., Chicago, Ill.
President-Elect: Dr. Wm. H. Evans, 24 Wick Ave., Youngstown, Ohio.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22, Pa.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Prof. Justo Alonso.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia, Pa.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Alden H. Miller.
Secretary-Treasurer: Dr. Victor Goodhill.
Chairman of Section on Ophthalmology: Dr. Dennis V. Smith.
Secretary of Section on Ophthalmology: Dr. Carroll McCoy.
Chairman of Section on Otolaryngology: Dr. Howard P. House.
Secretary of Section on Otolaryngology: Dr. Edwin Scobee.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Alfred Schattner, 115 E. 61st Street, New York 21, N. Y.
Secretary: Dr. Norman N. Smith, 291 Whitney Avenue, New Haven 11, Conn.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. G. M. Billings, Morganton, N. C.
Secretary and Treasurer: Dr. MacLean B. Leath, High Point, N. C.
Time and Place: Sept. 11-13, Hendersonville, N. C. Jointly with the South Carolina Society of Ophthalmology and Otolaryngology.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. David R. Higbee, 3245 4th St., San Diego, Calif.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco, Calif.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Thomas F. Furlong, Jr.
Vice-President: Dr. Harry P. Schenck.
Treasurer: Dr. William J. Hitschler.
Secretary: Dr. John J. O'Keefe.
Executive Committee: Dr. Valentine M. Miller, Dr. C. L. Jackson, Dr. George L. Whelan.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Alston Callahan, 908 S. 20th St., Birmingham, Ala.
Chairman-Elect: Dr. Francis LeJeune, Ochsner Clinic, New Orleans, La.
Vice-Chairman: Dr. Edwin Broyles, 1100 N. Charles St., Baltimore, Md.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: St. Louis, Mo., November 13-16, 1950.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Garnett P. Morison, Charles Town, W. V.
First Vice-President: Dr. Charles T. St. Clair, Jr., Bluefield, W. Va.
Second Vice-President: Dr. Arthur C. Chandler, Charleston, W. Va.
Secretary: Dr. Melvin W. McGehee, 425 Eleventh St., Huntington 1, W. Va.
Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
Directors: Dr. Eugene C. Hartman, Parkersburg, W. Va.; Dr. Ivan Fawcett, Wheeling, W. Va.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Victor M. Noubleau.
Secretary: Dr. Héctor R. Silva.
1o. Vocal: Dr. Salvador Mixco Pinto.
2o. Vocal: Dr. Daniel Alfredo Alfaro.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. Fernando Casadesus.
Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Sec. de Actas: Dr. Juan Berini.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Joao Penido Burnier.
First Secretary: Dr. Gabriel Porto.
Second Secretary: Dr. Roberto Barbosa.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. F. J. Monteiro Sales and Dr. Jose Martins Rocha.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. Juan Manuel Tato.
Vice-Presidente: Dr. Norberto Von Soubiron.
Secretario: Dr. Oreste E. Bergaglio.
Pro-Secretario: Dr. Carlos A. Gutierrez.
Tesorero: Dr. Vicente R. Carri.
Pro-Tesorero: Dr. Jorge Couzo.